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PATHOGENESIS AND ECOLOGY OF PANICLE BRANCH BLAST OF RICE PLANT

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I. Introduction

After war, the penetration of panicle branch blast of rice plant was widely noticed. For a long time, a serious panicle branch blast has been included in neck blast and a mild symptom had no designation. For example, Ito (1943) classified rice blast into neck, spikelet and glume blasts, but the panicle branch blast was not recorded. The panicle branch blast without penetrating the node at the neck of panicle was not treated as a neck blast unless more than 1/3 of panicle became white according to researcher's convention (Prevention of Plant Epidemic Section Plant Improvement Dept. Ministry of Agriculture and Forestry 1956). Thus, attention on panicle branch blast was very small. However, the panicle branch blast became important in warm area or warm autumn. Recently, the panicle branch blast has attracted attention at Kanto, Tohoku and Hokkaido regions.

The panicle branch blast refers simply to the case of invaded panicle branch, but when they are accompained by the desease of node at the neck of panicle, they are generally

referred as neck blast regardless of the existence of panicle branch blast. Therefore, the panicle branch blast indicates the case of invaded panicle branch without accompanying the desease of the node at the neck of panicle. In this paper, when panicle branch blast is clearly observed even if infection of node at the neck of panicle is present and the rice plant is not died, it is counted as neck blast and panicle branch blast. The desease extended to neck is counted as neck blast even if it originated from the panicle branch.

Researches on mechanism of panicle branch blast generation are very few and the infection is considered to occur simply at node of rachis or node of panicle branch (Sawada 1927, Yoshii 1936, Ito et al 1937, Kono 1954, Kono et al. 1960). Authors investigated the relation between evironmental change and rice blast, and observed the mechanism on the generation of panicle branch blast. Also we have observed importance of panicle branch blast while investigating the rice blast from various regions.

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II. Development of Desease

1. Observation by Inoculation

In order to observe the progress of panicle branch blast, observations are made by inoculating rice from pot cultivation.

a. Appearance of Branch Blast of Rice Plant, Specially from the Beginning of Desease to Death of Rice Plant.

(1) Experimental Method

Higashiyama No. 38 is cultivated during general period at outside in 1/2:000 a or 1/5,000 a Wagner Pot or 6 inch pot. After panicle has grown, the rice blast bacilli (1-2 bacilli strain) are inoculated by spray method, Pulp Method (Dasugi et al 1956), cotton method and injection method (Conidial liquid is injected into unripen glume) and the progress of desease is observed.

Designations of parts of panicle are based on Nagato (1950) and Matsushima (1956) and shown in Fig. 1.

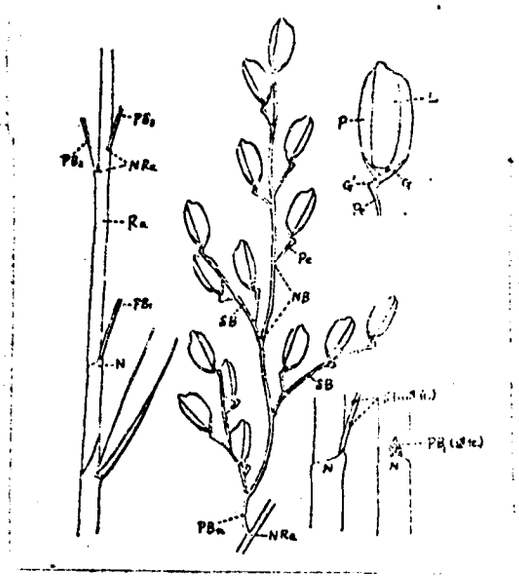

Fig. 1.—Designation of parts of panicle. N, node at the neck of panicle; Ra, rachis; NRa, node of rachis (the joints of the rachillae to the rachis); NB, node of panicle branch (rachilla); PB_1, the first primary panicle branch (rachilla); PB_2, the second primary panicle branch (rachilla); similarly, PB_3–PBn, primary branch No. 3–n; SB, secondary panicle branch or rachilla branch; P, palea; L, lemma; G, glume (empty glume); G′, subsidiary empty glume; Pe, pedicel.

(2) Experimental Results

In inoculated panicle, node at the neck of panicle, node of rachis, node of panicle branch, spikelet, primary or secondary panicle branch and other devoluted part of each organ are easily infected. The symptom of each infected part agrees with previous reports (Sawada 1927, Sumiya 1955, Kono et al 1960 and others). (Figure plate I).

We have observed that spikelet dies faster in case of

spikelet infection but when the lesion on the spikelet extends to the base direction and reach pedicel, the edges of the lesion produce smoewhat clear dark color-brownish purple color. (Figure Plate II, C. Fig. 2A). The lesion progresses to the direction of bottom part and reaches the node of panicle branch. When the lesion extends to the node of panicle branch, the spikelet dies (Fig. 2, B,C). The lesion extends to bottom part of rach's through the node and the death of panicle increases significantly accompanying these. The infection occurrs at distal parts and the lesion extends to bottom part i, e, base direction increasing the death of distal parts. These phenomena are designated as panicle death. Panicle death occurs when panicle branch or node of rachis are infected (Fig. 3). The death of distal parts increases remarkably in this case. Therefore, in a few days after development of desease, the desease symptoms of node of panicle branch, node of rachis and node at the neck of panicle are difficult to distinguish from symptoms of panicle death. When these nodes are penetrated by the panicle death, the lesion spreads rapidly and the color changes to clear as though the nodal parts are freshly infected. In spite of the rapid spreading of lesion to nodal parts, extension of lesion toward bottom part is temporarily stopped and the speed of panicle death is somewhat delayed. At initial period of desease, the typical symptoms of deseases are observed at each parts, and infected area can be judged from the location of penetration and mode of panicle death.

Based on these observations, the panicle branch blast is classified below and the relations between infection and panicle death are indicated.

Mode of Desease Development and Type of Panicle Branch Blast (include spikelet blast).

I. type (spikelet blast); Spikelets (palea, lemma and glume) are infected and each spikelet dies. A part or whole spikelet is penetrated, and the deseased part is usually grey-white. In many cases, the boundary of lesion is at part of bite or vascular bundle. Pedicel is healthy.

II-a type (secondary panicle branch blast originated from spikelet); Several spikelets die due to infection. Several grains on panicle branch die as a group and spikelet turns greyish white. Initial infection gives grey color in many cases. This type occurrs due to penetration on node of panicle branch by panicle death. The invaded part can be observed at the boundary between bottom part or upper part sometimes, of deseased area and healthy part.

II-b type (secondary panicle branch blast originated

from node of panicle branch); This occurrs due to the infection on the node of panicle branch and several spikelets die. At initial period of desease, the small lesions on the node of panicle branch give pinkish brown-brownish purple color. This resembles type II-a but no grey spikelets are observed.

III type (Primary panicle branch blast); This occurrs due to infection of spikelet or node of panicle branch and all the primary panicle branches die. The spikelets on primary panicle branches die and turn to greyish white color. The edges of lesions by panicle death can be observed near bottom part of primary panicle branch. The node of rachis is healthy.

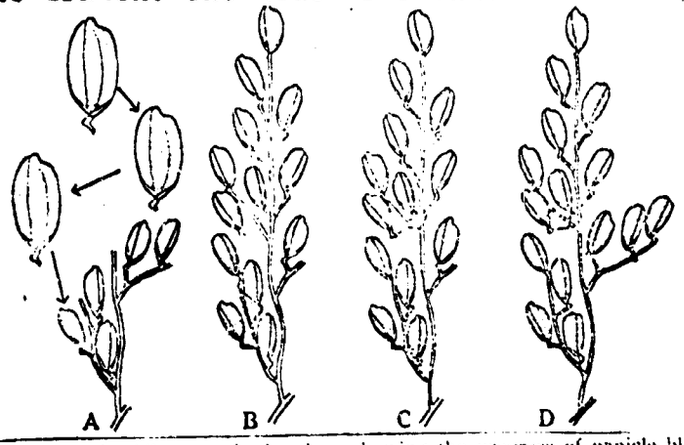

Fig. 2.—Semidiagramatic drawings showing the progress of panicle blast (B—D) from a spikelet blast (A).

A. The whole spikelet changes to greyish white several days after the appearance of lesions on the hull. Type I (spikelet blast).
B. The lesion on the spikelet extends to the node of panicle branchlet through pedicel eventually causing the death of all distal parts including spikelets, Type II-a (secondary panicle branch blast or rachilla branch blast, started from the spikelet).
C. An early symptom of Type III (primary panicle branch blast).
D. All the parts of the panicle branch have been killed, and the lesions are extending further to the rachis. An early symptom of Type IV (rachis blast).

IV type (Rachis blast) ; This occurrs due to infection on node of rachis or death of apical part and several or one primary panicle branch die. At initial period of desease, only the infected primary panicle branch dies. This resembles type III but differs in penetration at node of rachis. At latter period of desease, apical part, the part of initial infection easily becomes obscure.

According to these types, type I corresponds to spikelet blast and glume blast. II-IV types are so-called panicle branch blast. In type II(secondary panicle branch blast) or III (Primary panicle branch blast), the node of panicle branch is penetrated and in type IV (rachis blast), the node of rachis is penetrated. These show the death from deseased part to distal

parts. During the period of material collection, the secondary panicle branch blast (II type) can be classified into the case originating from spikelet infection (secondary panicle branch blast originating from spikelet, II-a type) and the case originating from infection at node of panicle branch (secondary panicle branch blast originating from node of panicle branch, II-b type). Generally, these types advance from type I to type IV and progress to neck blast. Very rarely an irregular progress is seen.

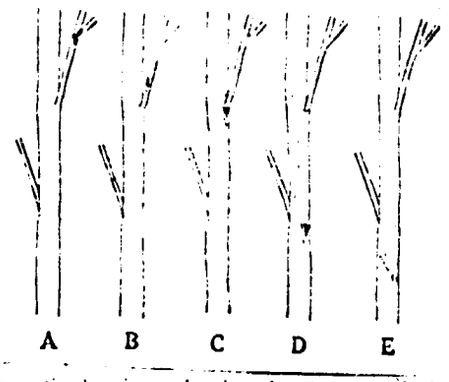

Fig. 3– Semi-diagramatic drawings showing the progress of the disease from a primary lesion at the node of a panicle branch (A) to panicle branch blast (B), then to rachis blast (C), then to neck blast (D, early stage and E, later stage).

A. Type II-b (secondary panicle branch blast started from the node of panicle branch).
B. Type III (primary panicle branch blast).
C. Type IV (an early stage of rachis blast).
D. An early stage of neck blast.
E. Neck blast. The lowest primary panicle branch is remaining alive for a while.

When node at the neck of panicle is infected after a few days of growth, the progress of lesion is slow and No. 1 primary panicle branch dies before the lesion extends to node at the neck of panicle. In spite of healthy No. 2-5 primary panicle branch, apical part of No. 6 panicle branch dies. Because primary panicle branch adheres at 2/5 opening (Nagai 1949, Matsushima et al 1956), No. 1 and 6 primary panicle branches are arranged on same vascular bundle and blast bacilli advance rapidly through this vascular bundle. At any rate, due to progress of panicle death, it is difficult to know initial infected area by these types long time after the desease has developed.

The phenomenon of "panicle death" can easily be reproduced by method of injection on glume in addition to pulp method. It is generally observed in spray inoculation method.

b. Effects of Infected Parts of Panicle on Death of Panicle Branch.

Since we know that the panicle branch is penetrated from infection of node of rachis, node of panicle branch and

spikelet, the effects of infection of each part on panicle branch blast are investigated.

(1) Experimental Method

Norin No. 1 and Huzisaka No. 5 are used. In 1/5,000a Wagner Pot, 4 Kg of rice paddy soil from Saitamaken Atanomachi, 10 g of ammonium sulfate, 15 g of calcium phosphate and 1 g of potassium sulfate are added. One stock(4 pieces in one stock) is planted in one pot, and cultiveted outdoor since June 20. Ammonium sulfate 5 g are given in early period. As three inoculations classification, inoculation at panicle completion period (18/VIII), inoculation at 5 days after panicle completion (23/VIII), and inoculation at 10 days after panicle completion (28/VIII) are established. Blast bacilli (P-2 bacilli strain) are inoculated by spray method and kept for 36 hours in green house with 26-27°C temperature. Then, they are left in glass room (26°C, 50-70 % humidity) and development of desease is observed. Numbers are placed on each stem and the relative locations of primary and secondary panicle branch and each spikelet are marked. Infected parts and progress of lesion are recorded periodically and the desease history is clearly kept. Seven stocks in seven pots are used for one classification and one among them is a standard pot without inoculation.

Infection number is indicated by counting the deseased spots in 50 plants until 15 days after inoculation and converting to number per one plant. The degree of panicle death is indicated by the degree of panicle branch desease on 21 days after inoculation. The white stem due to desease of node at the neck of panicle was excluded from the experiment. When panicle deathes originated from spikelet infection or infection at node of panicle branch and also due to infection of bottom part of rachis node appear as double symptoms, they are treated as infection on node of rachis. Panicle deathes by double infections of spickelet and node of panicle branch are similarly treated and the infection of distal parts are preferentially treated.

(1)	(2)	(1)	(2)
1~10	0.1	51~60	0.6
11~20	0.2	61~70	0.7
21~30	0.3	71~80	0.8
31~40	0.4	81~90	0.9
41~50	0.5	91~100	1.0

1. Rate of spikelet desease
2. Value index number

The degree of panicle branch desease is separated into ten steps of 0.1-1.0 from the proportion of deseased spickelets against total number of spickelets. The value index number is as follows. The degree of panicle branch desease is obtained by following equation.

$$\text{Degree of panicle branch desease} = \frac{\sum(\text{degree of primary panicle branch desease})}{\text{Total number of primary panicle branch}} \times 100(\%)$$

$$\text{Degree of panicle branch desease per one infected part (degree of lesion expansion)} = \frac{\Sigma(\text{degree of primary panicle branch desease})}{\text{Number of infected area}}$$

(2) Experimental Results

The results are shown in table 1-3. Through experiments, the desease is not observed in rice plant without inoculation. Table 1 shows the number of infected areas per one panicle. According to these, (1) infection on node of rachis and panicle branch is very small and infection on node at the neck of panicle is medium whereas infection of spikelets is very large. When node of rachis, node of panicle branch and actual number of spikelet (see figure 1, Morinaga et al 1943) are considered and compared, infection of spikelets is large between spikelets at node of rachis and node of panicle branch or between spikelet and node of rachis.

1)	5)	7)	8)	9)	10)	11)
2)	6)	4.6	0.2	0.2	0.55	
		6.2	0.2	0.2	0.55	
3)	6)	1.4	.1	0.1	0.26	
		2.7	0.2	0.1	0.16	
4)	6)	2.3	0.01	0.01	0.19	
		2.6	0.3	0.3	0.21	

12) 1)
2) 1956.

1. Inoculation period
2. Panicle completion period
3. 5 days after panicle completion
4. 10 days after panicle completion
5. Kinds
6. Norin No. 1 Huzisaka No. 5
7. Number of infected areas per one panicle
8. Spikelet infection
9. Infection at node of panicle branch
10. Infection at node of rachis
11. Infection of node at the neck of panicle
12. Remark 1). Inoculation at panicle completion period.
2). Experiment was conducted at Nishi Keharn (Agricultural Research Inst.) Tokyo in 1956.

Also, infection is severe on node at the neck of panicle, compared with other parts. 2) When infection of spikelets is considered according to inoculation period, the infection is severe in panicle completion period. Infection is rather small on 5 days and 10 days after panicle completion, and no significant difference between these two periods is observed. Among the kinds of panicles, Huzisaka No. 5 is large in either inoculation periods. 3) No significant difference between nodes of panicle branch and rachis is observed in these inoculation periods. Norin No. 1 had the most severe infection in panicle completion period but infection reduced rapidly on 5 days after panicle completion. Huzisaka No. 5 had the most infection between 5 and 10 days after panicle completion. (Huzisaka No 5 seemed to have more panicle branch blasts at Agricultural Station).
4) Infection on node at the neck of panicle is high in panicle completion period and reduces to half between 5 days and 10 days after panicle completion. The difference between the kinds of panicles is not clear.

Table 2

Degree of Panicle Branch Desease According to Regions of Panicle Branch Blast Infection.

1)		5) 品種	6) 穂数	7) 同一次枝梗	8) 項目	9) 穎感染	10) 枝梗苗感染	11) 穂軸苗感染	12) 其他の感染	13) 合計
2)	出穂期	農林1号	29	203	14) (一次枝梗罹病度)	58.2	10.0	15.0	0.8	84.0
					枝梗罹病度%	28.7	4.9	7.4	0.4	41.4
					同上比率	69	12	18	1	100
		藤坂5号	29	254	14) Σ(一次枝梗罹病度)	80.9	8.9	39.0	33.0	161.8
					枝梗罹病度%	31.9	3.5	15.4	13.0	63.7
					同上比率	50	6	24	20	100
3)	穂揃5日後	農林1号	52	355	14) (Σ(一次枝梗罹病度)	47.4	7.8	0.0	5.4	60.6
					枝梗罹病度%	13.4	2.2	0.0	1.5	17.1
					同上比率	78	13	0	9	100
		藤坂5号	30	243	14) (Σ(一次枝梗罹病度)	24.0	7.0	54.0	9.9	94.9
					枝梗罹病度%	9.9	2.9	22.2	4.1	39.1
					同上比率	25	7	57	10	100
4)	穂揃10日後	農林1号	32	223	14) (Σ(一次枝梗罹病度)	21.0	1.0	9.0	1.5	32.5
					枝梗罹病度%	9.4	0.4	4.0	0.7	14.6
					同上比率	65	3	28	5	100
		藤坂5号	27	230	14) (Σ(一次枝梗罹病度)	16.1	9.0	43.0	2.7	70.8
					枝梗罹病度%	7.0	3.9	18.7	1.2	30.8
					同上比率	22	13	61	4	100

1. Inoculation period
2. Panicle completion period {Norin No. 1 / Huzisaka No. 5
3. 5 days after panicle completion {Norin No. 1 / Huzisaka No. 5
4. 10 days after panicle completion {Norin No. 1 / Huzisaka No. 5
5. Kinds
6. Number of panicles
7. Number of primary panicle branches
8. Items
9. Spikelet infection
10. Infection on node of panicle branch
11. Infection on node of rachis.
12. Other infections.
13. Total
14. {Σ (Degree of primary panicle branch desease) / Degree of panicle branch desease (%) / Ratio of panicle branch desease

Table 2 shows the effects of each infected area on degree of desease. The following can be said from the table. 1) Taking the example of Norin No. 1 for panicle completion period, 28.7 % among total 41.4 % of panicle branch desease was spikelet infection and panicle branch desease ratio is 69 %. The degrees of panicle branch desease due to infection of node of panicle branch and node of rachis are 4.9 % and 7.4 % respectively. The ratio is 12 % and 18 % respectively. In other words, panicle death due to spikelet infection is very large and panicle death due to infection of node of panicle branch or node of rachis is very small. This relationship still hold at 5 days and 10 days after panicle completion. 2). Huzisaka No. 5 had almost the same relationship as Norin No. 1. However, on 5 days and 10 days after panicle completion, the degree of panicle branch desease due to infection of node of rachis increase and the desease due to spikelet infection decreased. Therefore, the desease of node of rachis increases. 3). In comparison with Norin No. 1 Huzisaka No. 5 has higher degree of panicle branch desease. This is due to high rate of desease on node of rachis in Huzisaka No. 5.

Table 3

Degree of Panicle Branch Desease per one Infected Area of Panicle Branch Blast (Degree of Lesion Expansion)

1)	5)	6) 穂数	7) 同一次枝梗数	8) 項目	9) 穎花感染	10) 枝梗節感染	11) 穂軸節感染
2)	農林1号	29	203	12) (Σ(一次枝梗罹病度)	58.2	10.0	15.0
				感染筒所数	78	10	4
				病斑拡大度	0.7	1.0	3.8
	越路5号	29	251	12) (Σ(一次枝梗罹病度)	80.9	8.9	39.0
				感染筒所数	105	9	7
				病斑拡大度	0.8	1.0	5.6
3)	農林1号	52	355	12) (Σ(一次枝梗罹病度)	47.4	7.8	0.0
				感染筒所数	126	8	0
				病斑拡大度	0.4	1.0	--
	越路5号	30	243	12) (Σ(一次枝梗罹病度)	21.0	7.0	51.0
				感染筒所数	40	7	8
				病斑拡大度	0.6	1.0	6.8
4)	農林1号	32	223	12) (Σ(一次枝梗罹病度)	21.0	1.0	9.0
				感染筒所数	38	1	4
				病斑拡大度	0.6	1.0	2.3
	越路5号	27	230	12) (Σ(一次枝梗罹病度)	16.1	9.0	45.0
				感染筒所数	40	10	8
				病斑拡大度	0.4	0.9	5.4

1. Inoculation period
2. Panicle completion period {Norin No. 1
   {Huzisaka No. 5
3. 5 days after panicle completion {Norin No. 1
   {Huzisaka No. 5
4. 10 days after panicle completion {Norin No. 1
   {Huzisaka No. 5
5. Kinds
6. Number of panicle
7. Number of primary panicle branch
8. Items
9. Spikelet infection
10 Infection of node of panicle branch
11 Infection of node of rachis
12 (Σ (Degree of primary panicle branch desease)
   (Number of infected area
   (Degree of lesion expansion

Table 3 shows the effects of infection on expansion of lesion (rather damage). According to these, spikelet infection does not kill all the spikelets on primary panicle branch on 21 days after inoculation at 26°C, but infection of node of panicle

branch kills all the spikelet on panicle branch. The effects of infection on node of rachis are very large and one area of infection kills a few primary panicle branches. In panicles with shorter days of growth, expansion of lesion is faster when compared with panicles grown longer. Thus, the degree of panicle branch desease increases. Among the kinds of panicles, Huzisaka No. 5 has a larger expansion.

Above can be summarized as follows. The degree of panicle branch desease is different depending on the part of initial infection, number of infections and extent of ripening. The effect of infected part on the degree of panicle desease is large for node of rachis, medium for node of panicle branch and small for spikelet. Also, the effect on fresh panicle is larger than old one. Since the absolute amount of panicle death has a multiplicative relation with expansion of lesion and the number of infections, the degree of panicle branch desease is high even if the effect of spikelet desease is small. Therefore, the desease of distal parts is sometimes important.

2. Observation at Experimental Station

As mentioned above, the mode of developing panicle branch blast by inoculation seems to be peculiar phenomenon in experimental environment. In order to confirm this phenomenon, natural deseases are observed at Aichiken State Agricultural Experimental Station Inabashi Branch (Hokusetsurakukun Inetake-chyo, 1956), Naganoken State Agricultural Experimental Station Hoka Experimental Branch (Minamiyasukumokun Hokachyo, 1957) and near Okayamaken State Experimental Station (Okayamashi Hokuho, 1958).

a. Method of Observation

The progress of desease is examined by classifications based on changes of inoculated desease symtoms. The materials employed in experiments will be specified wherever appropriate.

b. Results

(1) Results on the materials from Naganoken State Agricultural Experimental Station Hoka Experimental Branch(1957)

The materials collected at this Station on Sept. 13, 1957 were investigated in detail. Four stocks are selected from four kinds(Chusei Ginga(durability against desease;strong), Norin No. 10 (medium) Norin No. 29 (weak) and Norin No. 30 (weak)) which have similar period of growth and the types of deseases are classified. The number and degree of panicle desease are compared for each type.

By observing closely desease symtoms of each parts, the mode of desease development and expansion of lesion can be easily compared with the results of above observations. The color of deseased part is greyish white initially in spikelet and agrees with the result of inoculation. Only pinkish brown-dark brown color or dark brownish purple color on panicle branch is sometimes observed. Also no clear boundary lines between healthy and deseased parts are obtained. The results of investigations are shown in table 4-6.

1) Development of panicle blast

Table 4 shows number of deseased spots per one panicle by each classifications.

Table 4

Development of Panicle Branch Blast by Classifications

1) 品種	6) 穂数	7) 型別の発病箇所数 I型 8) (穎イモチ)	II-a型 9) (枝梗原二次枝梗イモチ)	II-b型 10) (枝梗着生原二次枝梗イモチ)	III型 11) (一次枝梗イモチ)	IV型 12) (穂軸イモチ)	13) イモチ
2) 農林30号	71	37 (5.1)	233 (3.3)	0 (0.0)	16 (0.2)	36 (0.5)	32 (0.45)
3) 農林29号	56	339 (6.1)	25 (0.4)	18 (0.3)	5 (0.1)	12 (0.2)	25 (0.45)
4) 農林10号	79	240 (3.0)	63 (0.8)	3 (0.03)	5 (0.1)	3 (0.03)	51 (0.68)
5) 中生銀河	78	403 (5.2)	30 (0.4)	2 (0.02)	1 (0.01)	5 (0.1)	16 (0.21)

14) 備考 1) カッコ内は1穂当たりの発病箇所数で示す。
2) 1957年9月13日，長野県立農試豊科試験地で調査した。

1. Kinds
2. Norin no. 30
3. Norin no. 29
4. Norin no. 10
5. Chusei Ginga
6. Number of panicles
7. Number of deseased areas by classifications
8. I type, II-a type, II-b type, III type, IV type.
9. Spikelet blast
10. Secondary panicle branch blast originating from spikelet
11. Secondary panicle branch blast originating from panicle branch
12. Primary panicle branch blast.
13. Rachis blast
14. Neck blast

14. Remar 1). Parenthesis shows the number of deseased spots per panicle
2). Investigated at Naganoken State Agricultural Experimental Station Hoka Branch on Sept. 13, 1957

According to table 4, 1) spikelet blast(I type) is the largest, secondary panicle branch blast originating from spikelet (II-a type) and neck blast follow and secondary panicle branch originating from node of panicle branch (II-b type), primary panicle branch blast (originating from spikelet or node of panicle branch, III type), and rachis blast (originating from node of rachis IV type) are the smallest. 2) When actual numbers of spikelet, node of panicle branch, node of rachis and node at the neck of panicle per one panicle are considered, the largest infection is on node at the neck of panicle, next is spikelet infection and infection of node of rachis and nodeof panicle branch is the smallest. 3) Among the different kinds, infection is the largest in Norin No. 30 and in order of Norin No. 29 (little weak), Norin No. 10 (medium), Chusei Ginga (little strong).

2) Degree of panicle branch deseach by classification

Table 5 shows the degree of panicle branch desease by classifications.

Table 5

Degree of panicle branch desease by classifications

1) 品種	6) 穂数	7) 同一次枝梗数	8) 項目	10) I型	11) II型	12) III型	13) IV型	14) 小計	15)
2) 農林30号	71	665	9) Σ(一次枝梗病度)	31.8	0.0	16.0	163.0	210.8	0.0
			枝梗罹病度%	13.8	0.0	2.4	15.5	31.7	0.0
			同上比率	44	0	8	49	100	—
3) 農林29号	56	539	9) Σ(一次枝梗病度)	7.8	5.8	5.0	26.0	44.6	12.0
			枝梗罹病度%	1.4	1.1	0.9	4.8	8.3	2.2
			同上比率	17	13	11	58	100	—
4) 農林10号	79	672	9) Σ(一次枝梗病度)	21.2	1.3	5.0	3.0	33.5	16.0
			枝梗罹病度%	3.6	0.2	0.7	0.4	5.0	2.4
			同上比率	72	4	15	9	100	—
5) 中生銀坊	78	797	9) Σ(一次枝梗病度)	10.8	0.6	1.0	7.0	19.4	0.0
			枝梗罹病度%	1.4	0.1	0.1	0.9	2.4	0.0
			同上比率	56	3	5	36	100	—

1. Kinds
2. Norin no. 30
3. Norin no. 29
4. Norin no. 79
5. Chusei Ginga
6. Number of panicle
7. Number of primary panicle branch
8. Items
9. Σ (Degree of primary panicle branch desease)
   Degree of panicle branch desease (%)
   Ratio of panicle branch desease
10. II-a type, secondary panicle branch blast originating from spikelet
11. II-b type, secondary panicle branch blast originating from node of panicle branch
12. III type primary panicle branch blast
13. IV type, rachis blast
14. Subtotal
15. Neck blast

According to table 5, rachis blast (IV type) and secondary panicle branch blast originating from spikelet (II-a type) occupy most of panicle branch blast. Thus, rachis blast and secondary panicle branch blast originating from spikelet have the most effects on panicle branch death. 1) In Norin no. 30 and no. 29, the rachis blast is the largest and the ratioes of panicle branch desease are 49 % and 58 %. Secondary panicle branch blast originating from spikelet (II-a type) is the next. In the other two kinds having more resistant property, the effect of secondary panicle branch blast originating from spikelet (II-a type) is large. Secondary panicle branch blast originating from node of panicle branch (II-b type) and primary panicle branch blast (III type) have a small effect. 2) Next, on ratio of panicle branch desease, the secondary panicle branch blast (II-a type) from spikelet infection had the highest ratio and occupied more than half of panicle branch blast. Rachis blast (IV type) follows and the ratio of panicle branch blast originating from node of panicle branch (II-b type, III type) was small. 3) The degree of panicle branch desease was in the order of Norin no. 30>Norin no. 29>Norin no. 10>Chusei Ginga. 4) The degree of panicle branch desease originating from neck blast was very low. This probably does not result in panicle death due to short time of growth but in later time, the panicle death increases rapidly as the desease progresses.

3) Parts of initial infection and expansion of lesion

Table 6 shows expansion of lesion on each infected parts in terms of degree of panicle branch desease per part.

According to the results of table 6, expansion of lesion based on secondary panicle branch blast (II type) is small and expansion due to rachis blast (IV type) is large. Spikelet has a small effect on panicle death because it is on distal part, and rachis has a large effect on panicle death because it is on base part. The effects are also large in Norin No. 30 and small in Chusei Ginga.

Table 6

Degree of Panicle Branch Desease per one Infected Area of Panicle Branch Blast

1) 品種	6) 穂数	7) 同一次枝梗数	8) 項目	10) II-a型 (穎着原二次枝梗イモチ)	11) II-b型 (枝梗節着原二次枝梗イモチ)	12) III型 (一次枝梗イモチ)	13) IV型 (穂軸イモチ)
2) 農林30号	71	665	9) Σ(一次枝梗罹病程度)	91.8	0.0	16.0	103.0
			発病籾所数	255	0	16	36
			病斑拡大度	0.4	—	1.0	2.9
3) 農林29号	56	539	9) Σ(一次枝梗罹病程度)	7.8	5.8	5.0	26.0
			発病籾所数	23	18	5	12
			病斑拡大度	0.3	0.3	1.0	2.2
4) 農林10号	79	672	9) Σ(一次枝梗罹病程度)	24.2	1.3	5.0	3.0
			発病籾所数	63	3	5	3
			病斑拡大度	0.4	0.4	1.0	1.0
5) 中生銀河	78	797	9) Σ(一次枝梗罹病程度)	10.8	0.6	1.0	7.0
			発病籾所数	30	2	1	5
			病斑拡大度	0.4	0.3	1.0	1.4

1. Kinds
2. Norin no. 30
3. Norin no. 29
4. Norin no. 10
5. Chusei Ginga
6. Number of panicles
7. Number of primary panicle branch
8. Items
9. Σ (Degree of primary panicle branch desease)
   Number of deseased areas
   Expansion of lesions
10. II-a type, secondary panicle branch blast originating from spikelet
11. II-b type, secondary panicle branch blast originating from node of panicle branch
12. III type, primary panicle branch blast
13. IV type, rachis blast.

(2) Results of materials from Aichiken State Agricultural Experimental Station Inabashi Branch (1956) and Hokuho Okayamashi (1958)

Materials from Aichi and Okayama were investigated and the results are reported in this section. The materials from Hokuho Okayamashi were from private farm near State Agricultural Experimental Station. In 1958, blast desease was light in this area. The materials from Aichiken Inabashi were Kinnanpu (resistant property-mediocre) and Soseigyoku (weak) in ripening period of Sept. 22, 1956. Materials from Okayamashi were Asahi (weak) in initial yellow ripening period of Oct. 4, 1958.

1) Development of desease on each part of panicle.

In order to examine development of desease on each part of panicle, frequency of infection is expressed for one panicle as shown in table 7. According to table 7, the desease of spikelet are signigicant and specially in case of Aichi, more than 5.5-7.5 spots per one panicle show up. Desease of node of rachis and node of panicle branch is small. These are similar to the results of materials from Naganoken Hoka. Materials from Okayamashi Hokuho had significant number of glume deseases. In case of Okayama, the material is Asahi and considered as the same strain of Soseigyoku. Since the desease of glume is considered to occur at latter period of ripening time, the weather of that year seemed to have a large effect.

Table 7

Development of Blast Desease on Each Part of Panicle

1) 調査材料	4) 品種	5) 1穂当たり発病箇所数 6) 内・外穎	7) 内・外護穎	8) 護穎	9) 退化穎	10) 枝梗節	11) 穂軸節	12) 退化枝梗	13) 穂首節
2) 愛知	早生旭	0.12	3.40	1.67	0.28	0.52	1.10	0.25	0.18
	金南風	0.02	6.25	1.38	0.07	0.07	0.08	0.05	0.02
3) 岡山	朝日	2.02		2.21	—	0.89	0.21	—	0.19

14) 註 1) 早生旭, 金南風はそれぞれ60穂, 朝日は110穂について調査。
2) 愛知では1956年9月22日, 愛知県立農試稲橋分場発生予察田において調査。
3) 岡山では1958年10月4日, 岡山市北方農家圃場において調査。

1. Materials for investigation
2. Aichi { Soseigyoku / Kinnampu
3. Okayama Asahi
4. Kinds
5. Number of deseased spots per panicle
6. Palea and lemma
7. Inner and outer glume
8. Glume

9. Devoluted spikelet
10. Node of panicle branch
11. Node of rachis
12. Devoluted panicle branch
13. Node at the neck of panicle
14. Remarks (1) Investigated 60 panicles each of Soseigyoku and Kinnampu
2) In Aichi, investigation is made on Sept. 22, 1956 at Aichiken State Agricultural Experimental Station Inabashi Branch
3) In Okayama, investigation is made on Oct. 4, 1958 at Okayama Hokuho farm.

2) Degree of Panicle Branch Desease on Parts of Initial Infection

In order to study effects of panicle branch blast on damage of initial infected areas, the degree of panicle branch blast on each infected parts was obtained and shown in table 8.

Table 8

Degree of Panicle Branch Desease on Parts of Initial Infection

1)		4)	5) 穂数	6) 同決枝梗数	7) 初発病部位別の枝梗罹病度 %					
					8) 穂	9) 枝梗	10) 穂軸	11) その他	12) 基部	13) 合計
2) 愛知		早生旭	60	465	4.8	1.2	0.9	0.9	0.2	8.0
		金南風	60	486	3.9	0.0	0.0	0.1	0.0	4.0
3) 岡山		朝日	110	1,028	5.6	3.7	6.1	—	9.9	25.3

1. Materials for investigation
2. Aichi { Soseigyoku / Kinnampu
3. Okayama Asahi
4. Kinds
5. Number of panicles
6. Number of primary panicle branch
7. Degree of panicle branch desease by deseased parts
8. Spikelet
9. Node of panicle branch
10. Node of rachis
11. Other
12. Node at the neck of panicle
13. Total

According to table 8, the degree of panicle branch desease of both kinds from Aichiken Inabashi is more than half. In case of Okayama, effect of neck blast is large and the desease of node of rachis and spikelet is the next in order of decreasing effect. Desease of node of panicle effect of spikelet desease on panicle branch desease is known to be large.

3) Speed of Panicle death from the deseased part

Since the death of deseased part in panicle branch blast is important as mentioned above, the speed of death by using Asahi is examined at Okayamaken State Agricultural Experimental Station, and inoculated rice (P-2 strain) and naturally deseased rice (experimental station farm) were also used in measurements. Thus, the deseased parts and dates were preexamined and the length of dead panicle is examined on end of October. The results are shown in table 9.

According to table 9, the length of dead panicle reaches 2-3 cm in one month after the development of desease. It is particularly significant that the length of dead panicle was not short when measured in short time after the development of desease. Also, for the same inoculation period, the length of dead panicle was longer for the desease developed earlier, indicating a somewhat faster growth. When the speed of panicle death is compared between infected parts, node of panicle branch was faster in the speed of death than spikelet. In spikelet, glume had faster speed of death than palea and lemma.

Table 9

Speed of Panicle Death from initially Deseased Parts

1)	2)	3)	4)			5)		6)		7)		8)	
			a)		b)	a)	b)	a)	b)	a)	b)	a)	b)
			mm			mm		mm		mm		mm	
9.12	9.23	10.22	41	20	16	31	28	27	12	32	0	—	
9.—	9.28	10.22	4	15	0		0	--	6	22	0	--	
9.12	10.3	10.22	1	15	0		0	--	2	21	0	—	
9.12	10.10	10.22	0	—	0		0	—	1	11	0	—	
9)	9.16	10.28	2	42	0	-	1	28	0	--	0	—	
	9.25	10.28	1	20	2	50	5	37	3	29	1	30	
	10.3	10.28	8	14	4	12	0	—	2	26	0	—	
	10.11	10.28	1	10	18	18	1	30	1	13	0	—	

10) 備考 1) [illegible] (1958年)
2) [illegible] (1958年)

1. Inoculation date
2. Date of initial desease development
3. Examined date

4. Palea and lemma a) Number of tests
b) Length of death
5. Glume a) Number of tests
b) Length of death
6. Inner and outer glume a) Number of tests
b) Length of death
7. Node of panicle branch a) Number of tests
b) Length of death
8. Node of rachis a) Number of test
b) Length of death
9. Naturally developed desease
10. Remark 1) Inoculation experiment is conducted at glass room of Okayamaken State Agricultural Experimental Station (1958)
2) Naturally developed desease was examined at farm of Okayamaken State Agricultural Experimental Stations (1958).

This is not unusual since the infected parts are different. In case of glume infection, mycelia reach to pedicel through small rachis, base part of glume and subsidiary empty glume. The small rachis operates similar to node of panicle branch and delays the extension of lesion toward bottom part. It is also considered to require longer time for mycelia to reach pedicel. Therefore, the death of spikelet seems to be somethat slow compared with panicle branch because there are many parts corresponding to nodes in same length.

(3) Summary

Status of desease on three regions is more or less the same. The panicle branch desease based on spikelet desease is considerable and panicle desease due to desease of node of rachis is about the same as spikelet. Panicle desease due to the desease of panicle branch is unexpectedly small. The importance of each parts and kinds is more or less different depending on period of investigation. Expansion of lesion from deseased parts is observed and the length of died panicle reaches 2-3 cm in one month. Natural panicle death is considered to occur similarly. Expansion of lesion is small for spikelet infection, medium for infection of node of panicle branch and large for infection of node of rachis. And it is small for resistant kind and large for succeptible kinds. Above facts are entirely similar to the results obtained from inoculation mehtod and the node of panicle branch blast agrees with observations of inoculation experiments.

III. Anatomical Observations of Spikelet Infection

In previous section, it was experimentally proved

that lesion of spikelet blast extends to bottom part of panicle and the desease is commonly observed in farm. As a first of oecological explanation of panicle branch blast, spikelet desease in farm, penetration of blast bacilli in spikelet and the activity at later time are of interest.

Designation of spikelet blast is rather old and Hayane (1901), Sawada (1927) and Furihara (1928) proved importance of spikelet desease experimentally. Uzi et al stressed the importance of direct damage by spikelet desease and seed infection. However, they did not consider the development of spikelet blast, and no further research on this subject has been reported. We have, therefore, made an experimental observation on this point.

1. Materials and Method

a) Natural Development of Blast Desease on Parts of Spikelet

On Sept. 22, 1956, Norin no22, Kinnanpu, Matsu-18, Homarekin and Ginga from late planting are examined at Aichiken State Agricultural Experimental Station Inabashi Branch. These correspond to initial period of desease. The existence of desease is comparatively examined on palea, lemma and glume respectively and also front, midile and bottom part of palea and lemma, from 10 panicles of each kind.

b) Anatomical view of natural spikelet desease

Since it is necessary to know the characterestics structure of glume and its parts from pathological and anatomical standpoints before observing penetration of spikelet blast bacilli, we have observed the pot cultivated rice at Nishikehara Kitaku Tokyo.

Among the materials having naturally developed spikelet desease from Aichi State Agricultural Experimental Station Inabashi Branch, Homarekin and Ginga are investigated. Relatively young spikelets are used but in case of ripened spikelet, the grains are removed before fixation and the subsequent operation is simplified. The materials are fixed with formalin, acetic acid and alcohol solution. Silicic acid is extracted with hydrogen flouride water and parafin is used for storing. Pieces of about 15μ thick are cut off and stained with Pianese III-b or Stoughton solution (Nishiyama 1952).

c) Penetration and spread of spikelet blast bacilli by Inoculation

Employing not-cultivated Aichigyoku from Nishikehara Kitaku Tokyo (Agricultural Technology Research Institute), the experiment is conducted with young spikelet to reduce the danger of natural infection. The first upper node of fully grown panicle is cut off and a part of leaf is removed. In order to expose apical part to bottom part, the surrounding spikelet is removed. Panicle is arranged in such a way that spikelet lies horizontally and blast bacilli (P-2 strain) are inoculated by spray method. Thus, the spikelets remain on panicle and the side of palea and lemma is inoculated. After inoculation, they are left in room at 26-27°C and taken out at 24, 48 and 72 hours. A part is settled in chromic acid, hypochromic acid and alcohol solution and the other part is settled in formalin, acetic acid and alcohol solution. After extraction of silicic acid and storage in paraffin, about 10 μ thick pieces are cut horigontally and vertically and observed after double staining with Safranin and Gentian violet.

2. Experimental Results

a. Deseased parts of naturally developed spikelet blast

The results are shown in table 10 and 11. According to table 10, total number of deseased spikelet on 50 panicles from 5 kinds was 1,292. 49 % of them had lemma penetration, 6 % palea penetration, 4 % glume penetration : 5 % both palea and lemma penetration and 36 % total penetration of palea, lemma and glume. When 628 spikelets having lesion on lemma are examined on apical part, center part and base part respectively (table 11), 71 % of them had tip, middle and base parts penetrated and 22 % had only tip part penetrated. Middle and base parts had very few desease and only 4 % and 3 % respectively.

Table 10

Appearance of Blast Desease Symptom on Each Parts of Spikelet.

1) 品種	8) 穂数	10) 内頴	11) 半頴	12) 護頴	13) 内・外頴	14) 内・半・護頴	15) 合計
2) 農林22号	10	26 (6)	192 (45)	10 (2)	10 (2)	186 (44)	424 (100)
3) 金南風	10	23 (6)	316 (52)	11 (3)	43 (10)	119 (29)	412 (100)
4) 来一	10	13 (5)	138 (48)	27 (9)	14 (5)	96 (33)	288 (100)
5) 銀河	10	5 (5)	16 (41)	1 (1)	3 (3)	56 (51)	111 (100)
6) 五葉糯	10	7 (12)	36 (63)	5 (9)	0 (0)	9 (16)	57 (100)
7) 合計	50	76 (6)	628 (49)	51 (4)	70 (5)	466 (36)	1,292 (100)

16) 備考 1) カッコ内は構成比を示す。
2) 1956年9月23日、愛知県農業試験場農業技術研究所において調査した。

1. Kinds
2. Norin no. 22
3. Kinnampu
4. Matsu -18
5. Ginga
6. Homarekin
7. Total
8. Number of panicles
9. Number of deseased spikelets by parts
10. Palea
11. Lemma
12. Glume
13. Palea and lemma
14. Inner and outer glume
15. Total
16. Remark 1) Parethesis indicates index ratio
2) Investigations are made on Sept. 22, 1956 at late plating farm of Aichi State Agricultural Experimental Station Inabashi Branch

Table 11

Appearance of Blast Desease Symptom on Each Parts of Lemma

1) 品種	3) 穂数	9) 籾位別での発病籾数 10) 先端部	11) 中位部	12) 基部	13) 先中基部	14)
2) 農林22号	10	28 (15)	2 (1)	3 (2)	159 (83)	192 (100)
3) 金南風	10	48 (22)	6 (3)	7 (3)	155 (72)	216 (100)
4) 来一 18	10	43 (31)	13 (9)	4 (3)	78 (57)	138 (100)
5) 銀河	10	3 (7)	0 (0)	1 (2)	42 (91)	46 (100)
6) 日本晴	10	16 (11)	1 (1)	4 (11)	15 (12)	36 (100)
7) 合計	50	138 (22)	22 (4)	19 (3)	449 (71)	628 (100)

15) 備考 1) カッコ内は指数比を示す。

1. Kinds
2. Norin no. 22
3. Kinnampu
4. Matsu-18
5. Ginga
6. Homarekin
7. Total
8. Number of panicles
9. Number of deseased spikelets by parts
10. Tip part
11. Middle part
12. Base part
13. Tip, middle and base part
14. Total
15. Remark 1) Parethesis indicates index ratio

More than half of deseased spikelets, whose tip, middle and base parts are penetrated, had the lesion spread through the middle part of spikelet (figure plate II, B_4 state). A part had penetration near base part. (figure plate II, A_3).

We have observed in other experiment that the desease of lemma was significant when Norin no. 1, Fuzisaka no. 5, Aichi-voku and Higashiyama no. 33 are inoculated at an early period. Thus, lesion is likely to form the lemma of spikelet and the desease seems to start near the region of hair in many cases.

The death of spikelet tip and phenomenon of greyish white coloration can occur from a mechanical damage of rachis or panicle branch. In this case, pedicel part of spikelet dies from dearth of water and turns white from the tip. In death due to dearth, part above the damaged spot of spikelet loses life and turns to white color from the tip of palea and lemma at the same time. This symptom is similar to the death of panicle branch due to penetration of panicle branch and rachis., but is different from those forming the lesion of death directly. In spikelet death due to penetration of rachis by blast bacilli, the color change into purplish brown is perceived first. When the base part of spikelet is penetrated, the change of color into greyish white, as mentioned above, was not observed. When the blast bacilli penetrate middle-base part of spikelet, early desease symptom becomes a partial lesion around the penetrated area.

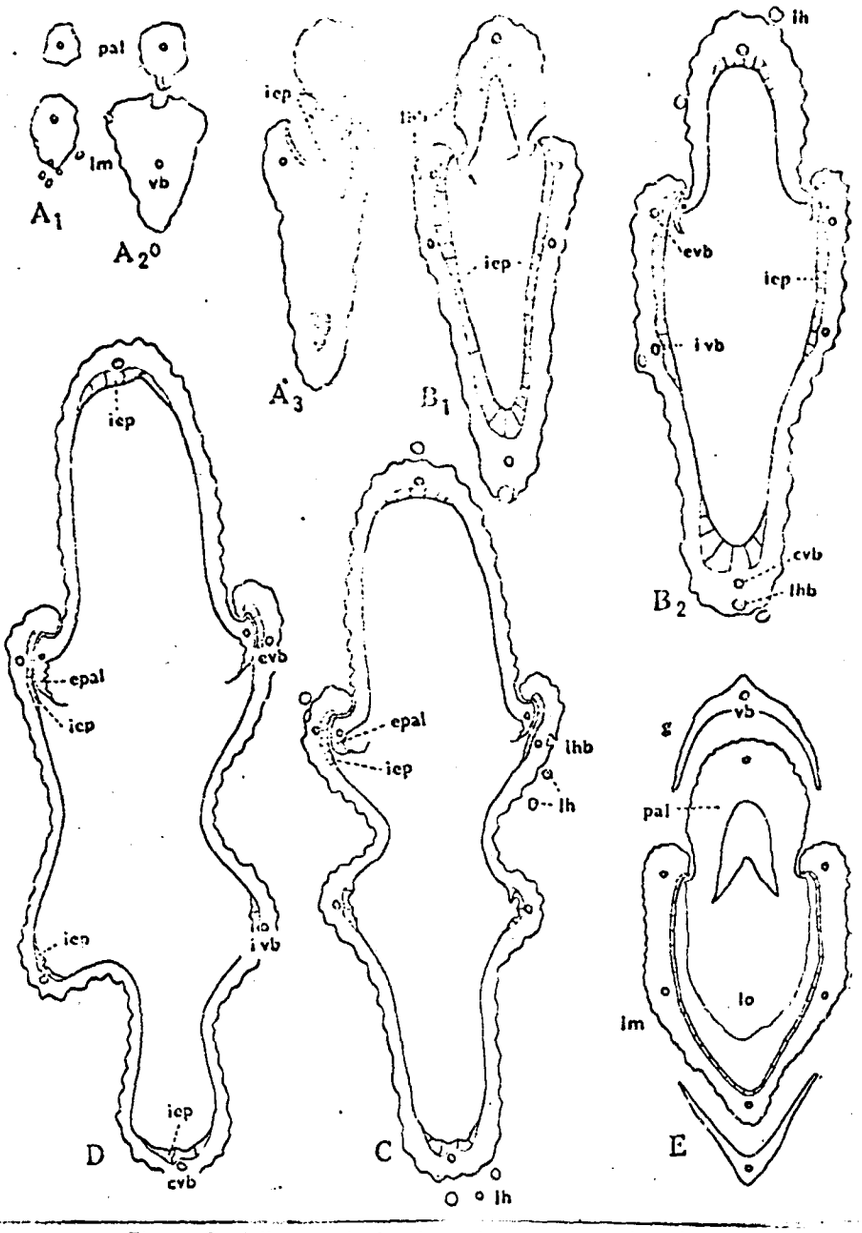

Fig. 4.—Semi-diagramatic drawings of transverse section of young spikelet.

$A_1 \sim A_3$. Tip of a spikelet. Contact of palea and lemma is incomplete and inner epidermis is half exposed.

$B_1 \sim B_3$. Shoulder of spikelet. Contact of palea and lemma began to be complete, and inner epidermis markedly thickens, especially in the area between two lateral veins.

C~D. Middle of a spikelet. The inner epidermis thickens in the vicinity of veins only. Cell walls at the periphery of palea thickens.

E. Basal part of a spikelet. The inner epidermis does not especially thicken.

pal, palea; lm, lemma; vb, vascular bundle; evb, external lateral vascular bundle; ivb, internal (lateral) vascular bundle; cvb, midrib; iep, inner epidermis; lh, large hair; lhb, bottom of large hair; epal, edge of palea; g, glume (empty glume); lo, lodicule; ov, ovary.

b. Texture of Glume from the Standpoint of Pathological Anatomy

(1) Hair

On surface of glume there are a small hair of two similar size cells and a large hair of single cell. Small hair of two cells forms on surface of cells. (Nishimon et al 1962) and the arrangement is regular. The shape resembles condition of micrococcus nucleus (figure plate III, A, B). Side view of this small hair is circular and slightly larger than appressoria of blast bacilli. When base part of cell surface is entrapped, it is easy to confuse with early period of bacilli penetration. Hair of large cell (figure plate III, C,D) can be observed with naked eye. Tip of hair has a narrow cylindrical shape and it grows on vascular bundles. Histologically, they originate on surface and the bottom part contact the lower surface. The hair seems to be easily damaged by spikelet friction. If bacilli penetrate through damaged part, the resistance against these bacilli is small and they extend to lower surface easily.

(2) Vascular bundle

There are three vascular bundles in palea and five in lemma.(Fig. 4, B_2) In lemma, there are one vascular bundle on midrib and two on both side (internal vascular bundle and external vascular bundle). External vascular bundle runs along the contacting part of palea and lemma. In palea, one runs along contacting part with lemma, and is arranged as though protected against outer part of spikelet in lemma (Fig. 4,C,D). The spiral tube flows into front part of lemma (Fig. plate IV, A, Figure plate V,B,C,D) and seems to connect water hole (drainage organ).

(3) Stomata and water holes

Stomata (including water holes) can be seen on inner and outer surface of glume. Table 12 shows the relation between each part of glume and number of stomata.

Table 12

Number of Stomata (including water holes) on Surface of Glume

1) 籾の縦方向分割		2) 切片数	3) 内面表皮 a) 内頴側	3) 内面表皮 b) 外頴側	4) 外面表皮 a) 内頴側	4) 外面表皮 b) 外頴側
1	上部	欠 6)	−(−)	−( )	−(−)	−(−)
	中 5)	10	−(−)	−(−)	8 (0.8)	31 (3.1)
	下	15	0 (0)	0 (0)	9 (0.6)	25 (1.7)
2	上	17	4 (0.2)	1 (0.1)	0 (0)	12 (0.7)
	中 5)	16	5 (0.3)	11 (0.9)	0 (0)	4 (0.3)
	下	18	11 (0.6)	19 (1.1)	0 (0)	2 (0.1)
3	上	8	4 (0.5)	7 (0.9)	0 (0)	2 (0.3)
	中 5)	16	8 (0.5)	14 (0.9)	0 (0)	3 (0.2)
	下	16	2 (0.1)	4 (0.3)	0 (0)	0 (0)
4	上	13	4 (0.3)	5 (0.4)	0 (0)	1 (0.1)
	中 5)	15	3 (0.2)	3 (0.2)	0 (0)	2 (0.1)
	下	15	3 (0.2)	7 (0.5)	0 (0)	1 (0.1)
5	上	欠 6)	−(−)	−(−)	−(−)	−(−)
	中 5)	5	3 (0.6)	1 (0.2)	0 (0)	0 (0)
	下	2	0 (0)	0 (0)	0 (0)	0 (0)
6	上	4	0 (0)	1 (0.3)	0 (0)	0 (0)
	中 5)	4	1 (0.3)	0 (0)	0 (0)	0 (0)
	下	3	2 (0.7)	2 (0.7)	0 (0)	0 (0)
7	上	3	1 (0.3)	0 (0)	0 (0)	0 (0)
	中 5)	6	1 (0.2)	1 (0.2)	0 (0)	0 (0)
	下	4	0 (0)	1 (0.3)	0 (0)	0 (0)
8	上	9	3 (0.3)	5 (0.6)	0 (0)	0 (0)
	中 5)	4	1 (0.3)	2 (0.5)	0 (0)	0 (0)
	下	2	0 (0)	0 (0)	0 (0)	0 (0)
9	上	欠	−(−)	−(−)	−(−)	−(−)
	中 5)	欠 6)	−(−)	−(−)	−(−)	−(−)
	下	欠	−(−)	−(−)	−(−)	−(−)

1. Vertical separation of spikelet
2. Number of cut pieces
3. Inner surface a) palea
   b) lemma
4. Outer surface a) palea
   b) lemma
5. Upper part
   Middle part
   Lower part
6. None

Remark 1) Parethesis indicates per one piece
2) Spikelet is separated vertically into 10 and numbered front tip as 1 and base part 10. Each part is reseparated into 3 part of upper, middle and lower part.
3) None means absence of cut pieces.

According to table 12, number of stomata on outer surface is large in lemma and small in palea. Also stomata are thick on tip part of spikelet and rough on middle part. No stomata are observed on bottom part. Stomata on inner surface (figure plate V,A) are different from outer surface. They are observed from front part to base part of spikelet and upper part has more. Around stomate, a soft tissue is developed and provide the better conditions for bacilli penetration.

Structure of water hole resembles those on leaves (Mizuue 1956, Dabei et al 1960) and has a crumbled shape of stomata (figure plate V, B,C,D). These agree with the findings of Gishiva (1933) and Baker (1954) in other plant.

(4) Tissues

Glume consists of following order from outer part. Outer epidermis (epidermis). Lower tissue (epidermis lower tissue) Soft tissue layer and inner epidermis (inner epidermis or epidermis) (figure plate III, A, Figure plate V,A). Epidermis cells on external glume have a large shape and are regularly arranged layer. External part has wavy shape. Breda De Haan (1911) reported that silicic acid is accumulated on fat outer wall. Lower tissue consists of thick narrow membrane type three layers of fibrous surface (Breda De Haan 1911, Translated by Harashima), and is in position to protect lower soft tissue (figure plate V,A). Thus, the penetrated bacilli receive a resistance from membrane wall of fibrous surface before reaching a soft tissue and seemed to proceed in vertical direction where the obstacles are less.

The soft tissue cell is small and membrane wall is thin. Generally, they develop near vascular bundle and boundary of lemma.(figure plate IV,D).

Internal epidermis cells are very large and wall cells is very thin (figure V,A). Internal epidermis of young spikelet swells in the direction towards glume. The swelling is not very noticeable near the bottom part of glume but is quite noticeable on upper shoulder part of spikelet(Figure 4, Figure plate IV). On shoulder part of glume, the space between external and internal vascular bundles is specially thick (figure 4, B_2, Figure plate IV,B). Thick internal epidermis cells seems to contract in early period and lose original shape by pressure in spikelet. Internal epidermis cells near vascular bundles develop into large ones and contraction comes later. Inernal epidermis of lemma difficulty forms cell membrane walls and is different in its development from others (Figure plate IV, C,D). In many cases, spikelet swells without contraction. Breda De Haan (1911) and Juliano et al (1937) treated this as simply internal

epidermis without special record. Internal epidermis does not have chance of receiving direct attack of bacilli except during the blooming period since it is exposed to air only during the blooming period. Due to incomplete contact of spikelet tip, internal epidermis can be easily contacted to outside (Figure 4, A_3) and requires an attention on infection mechanism.

To summarize above, 1) numbers of stomata and vascular bundles in lemma are large compared with palea, and internal epidermis is thick. 2) Tip part of spikelet has a large number of stomata and drainage tissues are developed. Thickness of internal epidermis is remarkable. Number of stomata decreases gradually from middle part to base part. This part lacks drainage tissues and internal epidermis is thick. 3) Hair is thick on upper part of spikelet and outer part of vascular bundles. In the space between palea and lemma, hair is thick on tip part of lemma. Thus, hair , vascular bundles, number and arrangement of stomata and water holes or arrangement of soft tissure and thickness of internal epidermis are different depending on parts of glume. It is difficult to say that there is no relation between structural characteristic and bacilli penetration. Although there is no direct relationship, there seems to be some effects.

c. Anatomical View of Naturally Deseased Spikelet

When pieces of glume are stained, healthy tissues are green, deseased tissues pinkish brown~brown color and mycelia, conidophore and conidium pinkish brown-tea brown color by Pianese III-b staining solution. And by Stoughton solution, membrane tissues are green, cell membran yellow and bacilli pinkish brown-tea brown color.

When spikelets having lesions on upper half (Figure plate II, B_2 state) are considered, they are as follows. The variation in tissue desease is noticeable around center part of lesion and becomes lighter away from the center part. Palea without lesion is considered healthy. Tissues are destroyed at the center of deseased part and the destruction of tissues is severe on soft tissues and internal epidermis. Near the boundary of healthy and deseased parts, many soft tissues and internal epidermis cells are destroyed. In that case, external epidermis and lower tissues do not have many variations in desease. The variation in desease of internal epidermis near the boundary of lemma is noticeable and cells which appear healty externally had mycelia in many cases.

Conidophore and conidium formed deseased parts of external epidermis or internal epidermis of glume. On external

epidermis, they occur mainly on boundary of lesion tissue and healthy tissue and are easily observed on base part of large hair in single cell (figure plate VI).

Table 13

Parts of Spikelet and Spread of Mycelia (example 1)

1) 穂の位置	2) 内	3) 外		類	
	4) 外穎	内穎近 (a)	5) (a)と(b)の中間	6) 内穎	内穎近 (b)	7) 中軸	軸近
1 上部	欠9)	欠	欠	欠	欠	欠
1 中・8)	−	−	−	−	−	−
1 下・	−	−	−	−	−	−
2 上・	−	−	−	−	−	−
2 中・8)	−	+	+	+	−	−
2 下・	+	+	++	++	−	−
3 上・	+	++	+++	++	+	+
3 中・8)	+	+++	+++	+++	−	−
3 下・	+	+++	+++	++	+	+
4 上・	++	+++	++~+++	++~+++	(++)	(++)
4 中・8)	+	+++	++~+++	++~+++	+	+
4 下・	+	+++~++++	++~+++	+++	++	++
5 上・	欠9)	欠	欠	欠	欠	欠
5 中・8)	+	++	+++	+	+	+
5 下・	+	++	+++	−	−	−
6 上・	++	+~++	++~+++	(+++)	+	+
6 中・8)	+~++	+++	++	++	++	++~+++
6 下・	−	+~++	++	−	−	−
7 上・	(+)	+~++	(+++)	+	+	+
7 中・8)	−	++	+	(+)	−	−
7 下・	−	−	++	−	+	+
8 上・	−	++	++	−	+	+
8 中・8)	−	++	(++)	(+++)	−	−
8 下・	−	−	(++)	−	−	−
9 上・	欠9)	欠	欠	欠	欠	欠
9 中・8)	欠	欠	欠	欠	欠	欠
9 下・	欠	欠	欠	欠	欠	欠

1. Vertical separation spikelet
2. Palea
3. Lemma
4. Near external vascular bundle (a)
5. Space between (a) and (b)
6. Near internal vascular bundle (b)
7. Near midrib
8. Upper part
   Middle part
   Lower part
9. None

Remark 1) Degree of mycelial spread is indicated by following standard.

-: No mycelia ##; Many mycelia
+; Very few mycelia ###; Very many mycelia
#: Few mycelia (); Terminal type

2) Horizontal and vertical continuous pieces are examined. 3-4 pieces
3) Mycelia in tissures can be classified. Number of penetration was proportional to mycelial density in tissues.

In glume, formation of conidophore and conidium is observed on internal epidermis of vascular bundles or boundaries (figure plate VI,C).

Spread of mycelia in tissues is noticeable on vertical direction of spikelet but not significant on horizontal direction. This tendency is observed on soft tissue, internal epidermis and lower tissure of membrane, but is not clear on external epidermis. An example of mycelial spread in tissue near the boundary of lemma is given below.

Even when desease has progressed in lemma destroying the tissues and many conidia are formed on surface, the tissues in adjacent palea are healthy in many cases. This type of desease is commonly seen in a closely contacted state of lemma and palea. From these, it can be concluded that bacilli can not move smoothly from one side to other on contact part. The fact that movement of bacilli in horizontal direction is limited is probably due to thickening of tissues on outer edge of lemma and protrusion of thickened palea forming a separation wall (figure IV, C, D).

Table 14

Parts of Spikelet and Spread of Mycelia (Example 2)

1) 穎の縦方向分割		2) 内	3) 外			
			4) 外維管束近(a)	5) (a)と(b)の中間	6) 内維管束近(b)	7) 中肋近
1	上部	欠 8)	欠	欠	欠	欠
	中〃 9)	欠	欠 8)	欠	欠	欠
	下〃	欠	欠	欠 8)	欠	欠
2	上〃	欠	欠	欠	欠 8)	欠
	中〃 9)	欠	欠	欠	欠	欠 8)
	下〃	−	±	++	−	−
3	上〃	−	+~++	+	−	+
	中〃 9)	−	++	+	−	(++)
	下〃	−	++~+++	+~++	+~++	−
4	上〃	−	−	+	−	−
	中〃 9)	−	+	+	++~+++	−
	下〃	−	−	+	++	−
5	上〃	−	++~+++	+	++~+++	−
	中〃 9)	−	++	−	++	−
	下〃	−	++	−	++	+
6	上〃	−	++	−	+~++	−
	中〃 9)	+	++	−	−	−
	下〃	−	+~++	−	−	−
7	上〃	−	+	−	−	−
	中〃 9)	−	−	−	−	−
	下〃	−	−	−	−	−
8	上〃	−	−	−	−	−
	中〃 9)	−	−	−	−	−
	下〃	−	−	−	−	−
9	上〃	−	−	−	−	−
	中〃 9)	−	−	−	−	−
	下〃	−	−	−	−	−

1. Vertical separation of spikelet
2. Palea
3. Lemma
4. Near external vascular bundle (a)
5. Space between (a) and (b)
6. Near internal vascular bundle (b)
7. Near midrib
8. None
9. Upper part
   Middle part
   Lower part

Above facts indicate that bacilli penetration on upper part of spikelet occurrs near tip and mycelia spread to each tissue.. If conduct tissues were penetrated, the supply of water for distal parts is obstructed and the tip part of spikelet seemed to turn white grevish color and die.

d. Penetration and spread of spikelet blast bacilli by inoculation

Healthy tissues are stained to pinkish yellow and bacilli are stained to pinkish brown-brown color by double staining of Safranin and Gentian violet. Appressoria is observed on concave part rather than wavy corvex part on external epidermis in many cases. In this experiment, bacilli penetrate through thick membrane of external epidermis and no penetration through epidermis cell or aperture cell was found. However, penetration through open part of large hair cell near base part was observed (figure plate VII, D). This open state of hair is often seen regardless of bacilli pentration. It is not clear, however, whether the opening is due to cutting action or easily splitting structure.

A thin mycelium from appressoria is penetrated through surface membrane and mycelia that reached external epidermis cell become thick and spread. Mycelia, then, proceed to inside of glume tissue and the spread at lower tissue seems to be slow. The spread of mycelia is very rapid in soft tissue and internal epidermis. In the case of bacilli penetration through base part of hair, mycelial spread in the direction of front tip of hair is not observed but the spread in the direction of internal epidermis was very active.

The relation between the lapsed time after inoculation and mycelial spread is as follows. 1) Several epidermis cells are penetrated after inoculation. Some times, a small amount of mycelia is observed in lower tissues of thick membranes but is not observed in soft tissue and internal epidermis. 2) Mycelia become thick in lower tissues after 48 hours and a part of them is observed in soft tissue and internal epidermis. Mycelia in tissues can be classified by penetration unit. 3) Mycelia in each tissue become thick after 72 hours and spread rapidly, and classification by penetration unit is difficult.

Degree of bacilli penetration and spread and its relation with each part of spikelet are shown in table 13-15.

Table 13-15 show the results on each spikelet from upper part to base part in 48 hours after inoculation. Four or five pieces per part are examined and the state of highly frequent

Table 15

Spikelet Parts and Spread of Mycelia (Example 3)

1) 穂の上下方向分部	2) 内穎	3) 外穎 4) 外維管束附近	5) 外内維管束の中間	6) 内維管束附近	7) 中肋附近
1 上部	欠	欠	欠	欠	欠
1 中〃8)	欠	欠	欠	欠	欠
1 下〃	欠9)	欠9)	欠9)	欠 9)	欠9)
2 上〃	欠	欠	欠	欠	欠
2 中〃8)	欠	欠	欠	欠	欠
2 下〃	欠	欠	欠	欠	欠
3 上〃	欠	欠	欠	欠	欠
3 中〃8)	欠	欠	欠	欠	欠
3 下〃	欠	+	++~+++	++~+++	+~++
4 上〃	+	++~+++	+++~++++	+~++	+
4 中〃8)	−	++~+++	+++	++	(+++)
4 下〃	+	+	+++	+++	+
5 上〃	−	++++	+++	++	+
5 中〃8)	+~++	++++	++++	++~+++	+~++
5 下〃	++~+++	+	+	−	+
6 上〃	−	+	++	+	+
6 中〃8)	+~++	−	−	+	−
6 下〃	(++)	+	+	−	+~++
7 上〃	+~++	−	+	−	+~++
7 中〃8)	++	−	−	−	−
7 下〃	+	−	+	(+)	+
8 上〃	+	−	−	−	−
8 中〃8)	−	−	−	−	−
8 下〃	−	−	−	−	−
9 上〃	−	−	−	−	−
9 中〃8)	−	−	−	−	−
9 下〃	−	−	−	−	−

1. Vertical separation
2. Palea
3. Lemma
4. Near external vascular bundle (a)
5. Space between (a) and (b)
6. Near internal vascular bundle (b)
7. Near midrib
8. Upper part
   Middle part
   Lower part
9. None

occurrence are indicated for convenience, although the degree of spread is slightly different depending on pieces. The following can be said from these observation. 1) When palea and lemma are compared, mycelia are small in palea tissues but large in lemma. Mycelial penetration and spread are quite noticeable near external or internal vascular bundles, and are small in the space between them. Mycelial penetration and spread are also small in the space between palea and midrib. 2) When spikelets are examined on tip art, middle part and base part respectively, the penetration is large near middle part, and mycelia in tissues are thick. Although the appressoria formation is easily observed from middle part to base part, no penetration is seen in many cases and the penetrated part becomes terminal type (figure plate VIII, D). When bacilli could not penetrate, outer membrane of epidermis cell is stained to deep brown color. In terminal type, several epidermis cells are stained to pinkish brown and mycelial migration into adjacent cells was not observed despite the presence of relatively thick mycelial concentration. When mycelial spread is active and its movement is smooth (figure plate VIII, A), the cell membrane is not stained to deep brown, and the degree of mycelia staining is light. 3) Several cells are penetrated on external epidermis and mycelia proceed to neighboring cells gradually. Mycelia in cells of lower tissues are not thick but extended to vertical direction of spikelet. The spread of mycelia in soft tissues and internal epidermis is specially active and when mycelia reach a part of this tissues the mycelia concentration increases rapidly.

Penetration of glumes is not observed.

In summary, the blast bacilli penetrate through thick membrane of epidermis. Mycelia in lower tissues are easy to extend in vertical direction of spikelet and mycelial movement in soft tissues or internal epidermis spread in vertical and horizontal direction. Therefore, mycelia fill each tissue with a lapsed time after penetration. When palea and lemma are compared, penetration is large in lemma and mycelial spread is active in tissues. Among tip part, middle part and base part of spikelet, penetration is most in tip part, middle is slightly less and penetration is very hard in base part.

IV. Wound and Panicle Branch Blast*

As there has been designation of wind blast (Ito 1933), it is known that there is a close relationship between blast desease and wind. However, effects of artificial wind on rice blast and panicle branch blast are not investigated. We have, therefore, investigated to make causal relation between

wind and w ticle dam ge or blast clear.

This experiment is conducted at Hudo (Tani, 1952) of Agricultural Technology Research Institute (Nishikehara Kitaku Kyoto).

1. Experimental Method

Norin no. 1 and Higashiyama no. 38 are grown outdoor and treated with wind. Blast bacilli are inoculated afterwards. Experiments are conducted 4 times, and the following table shows the kinds of rice and others. Fertilizer administration is shown in chapter II.

1) 試験区	2) 品種	3) 播種月日	4) 移植月日	5) 使用ポットの大きさ a.	6) 出穂期 (月日)	7) 接種時期	8) 同左月日	9) 発病調査月日
I 10)	農林1号	5.13	6.20	1/5,000	8.15	出穂期 12)	8.15	9. 2
II 10)	農林1号	5.13	6.20	1/5,000	8.15	乳熟期 13)	8.23	9.10
III 10)	農林1号	5.13	6.29	1/2,000	8.20	出穂期12)	8.30	9.17
IV 11)	東山38号	4.22	5.28	1/2,000	8.30	出穂期12)	8.30	9.17

1. Experimental no.
2. Kinds
3. Planting date
4. Transplanting date
5. Size of pot (a)
6. Panicle formation period
7. Inoculation period
8. Inoculation date
9. Date of desease examination
10. Norin no. 1
11. Higashiyama no. 38
12. Panicle formation period
13. Ripening period.

The wind speed was in two steps of 12 m per second (B region) and 9 m per second (A region). First, fan for B point is adjusted to give wind speed of 12 m per second. And then, the position of wind speed of 9 m per second is determined by an anemometer and designated as A point. Thus, treatment of two different wind speeds can be conducted simultaneously by placing rice plant on each position. These relations are schematically shown in Fig. 5. Also, wind speed, room temperature and humidity during treatment are recorded. According to Viewfort wind force table, wind speed of 9 m per second corresponds to wind force of 5 and 12 m per second corresponds to

(* Content of this chapter has been reported in Kanto Higashiyama Virus Research Report 5, 5-6 (1958)).

wind force of 6 (Japan Agricultural Meteological Society 1954). According to an idiom, they correspond to a violent wind and a strong wind respectively (Miura 1948). The conditions of treatment are shown in table 16.

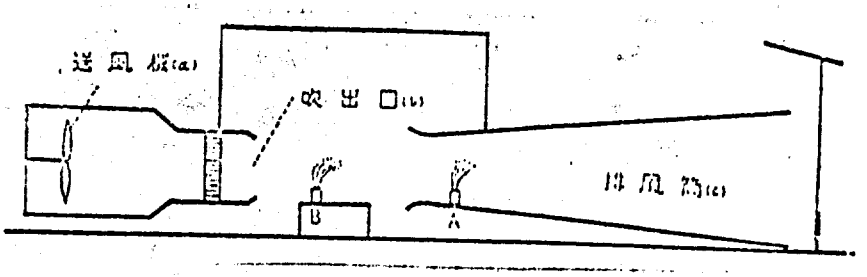

Fig. 5.—Showing a schematic drawing of the wind tunnel experiment. A and B, rice plant in pots; a, fan; b, blow-out; and c, exhaust.

Table 16

Conditions of Wind Treatment

1) 実験区	2) 品種	3) 風速 (m/sec) A区	B区	4) 時間	5) 処理月日	6) 処理時刻	7) 天候	8) 処理時室内の 9) 温度 °C	10) 相対湿度 %
I	農林1号	9.0~9.4	12.0~12.5	2.5	8.15	14.20~16.50	晴	27.0~28.0	74~77
II	11)農林1号	9.0	12.1~12.6	3.0	8.23	10.00~13.00	晴	33.0~33.4	48~54
III	農林1号	—	11.8~12.1	2.5	8.30	11.05~13.35	晴	26.8~29.8	47~65
IV	12)東山38号	—	11.8~12.1	2.5	8.30	11.05~13.35	晴	26.8~29.8	47~65

1. Experimental no.
2. Kinds
3. Wind speed (m/sec) A region B region
4. Time
5. Treatment date
6. Treatment time
7. Weather
8. Treatment room
9. Temperature
10. Humidity
11. Norin no. 11
12. Higashiyama no. 38
13. Clear

Within one hour after treatment, blast bacilli (P-2 strain) are inoculated and kept in a humid room of 26-28°C for 36 hours. Then, the materials for experiment II, III, IV are placed in glass room (26°C) and those for experiment I are placedoutdoor. Outdoor materials are brought into room when wind is strong in order to avoid panicle damage by wind. Degree of damage on

spikelet, panicle branch and leaf was observed right after treatment by staining with neutral red based on the method of Takahara et al (1955). After inoculation, observations are made without staining. Desease is examined on 18 days after inoculation according to classification of chapter II.

Since degree of panicle was large in Norin no. 1 during treatment of ripining period (Experiment II) and observation of panicle branch blast was difficult, investigation on desease of panicle was discontinued and only lesion on leaf was observed.

2. Experimental Results

As can be seen from table 16. the variation in the speed of wind during the treatment was less than 0.5 m in both A and B regions throughout experiments.

Leaves, rachis, panicle branch and spikelet are damaged by the treatment of artificial wind and the degree of damage was severe in 12 m per second wind speed (B region) and light in 9 m per second (A region). Exposure to 12 m per second wind for 2.5-3 hours resulted in splitting the leaf tips and the color of leaf turned to grevish green. Grevish green coloration is particularly apparent near the split part of leaf, but not noticeable near the center part. The coloration was very light on base part. Exposure to 9 m per second wind for 2.5-3 hours did not result in split leaves and the degree of damage was very light. Rachis, Panicle branch and spikelet are likely to be damaged on outside. Wound of rachis and panicle branch shows the line shape along the vascular bundles. The wound mainly occurs on half circumference of rachis and the other half away from wind direction has healthy appearance in many cases. This wound turns to brown color. Since half circumference of rachis is green and the upper and lower part of brown coloration is not clear unlike the lesion resulting from penetration of blast bacilli. it can be clearly distinguished from lesion.

A slightly swelled parts of spikelet surface or near the shoulder are easily wounded. This wound appears as spots in few days after treatment and has a clear edge of brown color. It is commonly an elliptical shape and as large as the lesion on desease of rice plant leaves. The central part has a grey color. These spots appear easily in region of 9 m wind speed and are easily found on old panicle. Bacilli are not observed on these spots. The spikelets turn to dark brown color in 2-3 days after treatment. Dark brown coloration is light in the region of 9 m wind speed and severe in the region of 12 m wind speed. The spikelets did not turn to

dark brown color but the part exposed to air had the dark brown coloration during the treatment. Young spikelets are easily wounded and the wounds turn to dark brown color. Old spikelets are difficultly wounded and the wounds are in spots. Degree of damage on rachis, panicle branch and small branch is opposite to the case of spikelet when viewed from growth days: Damage is light in initial growth period and heavy in ripening period. The old materials had severe damage.

From these observations, we note that young panicle has heavy damage in spikelet and old panicle has heavy damage in branch. The fact that old panicle branch is easily damaged indicates that silicification of panicle branch makes more susceptibel to wound and promote partial death due to the lack of ability to meet sudden change in moisture renewal by wind. In young panicle, spikelet surrounds outside of panicle branch. Thus, the degree of wound is high in spikelet and low in rachis.

Desease of leaves is as shown in table 17 and formation of S type lesion (Advancing type) was not observed. M type (standard discontinued type) and R type (brown spot type) lesions are small in region of 9 m per second wind speed and large in region of 12 m per second wind speed. When experiment I (treatment at initial growth period) and II (treatment at ripening period) are compared, it was lager in experiment I. This agrees with the fact that young leaves are more susceptible into leaf blast (Goto et al 1961). Without treatment, only few R type lesions are found in experiment II. Rice plant did not have desease without inoculation regardless of wind speed.

Table 17

Artificial Wind Treatment and Desease of Leaves (Higashiyama No. 38)

1) 実験区	2) 出穂後* 日数	3) 接種区分	4) 風速	5) 時間	6) 葉数	7) 1葉当たり病斑数 8) R型	M型	S型
I	0～2日 9)	接種	標準無処理	—	33	0.0	0.0	0.0
			A (9 m)	2.5	34	0.6	0.1	0.0
			B (12m)	2.5	39	2.0	2.8	0.0
		無接種	標準無処理	—	10	0.0	0.0	0.0
			A (9 m)	2.5	9	0.0	0.0	0.0
			B (12m)	2.5	8	0.0	0.0	0.0
II	8～11日 9)	接種	標準無処理	—	23	0.1	0.0	0.0
			A (9 m)	3	27	0.1	0.1	0.0
			B (12m)	3	23	0.9	0.6	0.0
		無接種	標準無処理	—	11	0.0	0.0	0.0
			A (9 m)	3	9	0.0	0.0	0.0
			B (12m)	3	9	0.0	0.0	0.0

1. Experimental no.
2. Growth time in days
3. Inoculation
4. Speed of wind
5. Time
6. Number of leaves
7. Number of lesions per leaf
8. I type, II type, III type, IV type, neck
9. Inoculation standard, no treatment
   - A (9m)
   - B (12 m)
   - No inoculation standard, no treatment
     - A(9 m)
     - B(12 m)
10. Note; Considering the difference in desease due to age of leaves, plant of same age group was investigated.

The lesions of leaves are formed at tip of leaves and concentrated near the split part. Desease was not found at center and base part of leaves. Desease is seen in the part of severe wound at tip of leaves.

Desease of panicle branch blast is shown in table 18. According to table 18, number of deseased areas per panicle by the types of panicle branch blast was very small in non-treated region (0 m wind speed), large in 9 m wind speed and very large in 12 m wind speed region.

Table 18

Treatment of Artificial Wind and Panicle Blast

1) 実験区別	2) 品種	3) 接種の有無	4) 風速 (m/sec)	5) 1穂当たり発病数 6) I型	II型	III型	IV型	首	7) 枝梗罹病度% 8) II型	III型	IV型	首
		+	0	0	0.7	0	0	0.49	4	0	0	13
I 9)	農林1号	+	9	0	1.3	0.3	0.1	0.50	8	4	1	0
		+	12	0	1.5	0.2	0.1	0.60	11	3	3	5
		−	0,9,12	0	0	0	0	0	0	0	0	0
		+	0	0.4	0.8	0.02	0	0.20	1	0	0	0
III 9)	農林1号	+	12	0.4	4.6	0.2	0.1	0.33	18	3	5	0
		−	0,12	0	0	0	0	0	0	0	0	0
		+	0	0.2	0.6	0	0.02	0.19	3	0	1	0
IV 10)	東山38号	+	12	0.1	2.1	0.1	0.3	0.23	7	1	14	0
		−	0,12	0	0	0	0	0	0	0	0	0

1. Experimental no.
2. Kinds
3. Existence of inoculation
4. Speed of wind (m/sec)
5. Number of desease per panicle
6. I type, II type, II type, IV type, neck
7. Degree of panicle branch desease (%)

8. II type, III type, IV type, neck
9. Norin no. 1
10. Higashiyama no. 38
Remark 1) 50 panicles per experiment.

IV type (rachis blast(, III type (primary panicle branch blast) and I type (spikelet blast) had small number of lesions whereas node of panicle branch or II type originating from spikelet has almost same tendency as the number of infections. The above facts indicate an agreement between degree of wound and desease of parts and panicle branch blast and express that many wounds panicle branch blast and express that many wounds result in many deseases.

Neck blast has an almost similar tendency as panicle branch blast. Neck blast in experiment I (Norin no. 1) for non-treated case resulted in many death in spite of the relatively few infections. It is not clear whether this is due to the difference in a small number of tests or other reasons. Variation in number of deseases was large in panicle branch blast and small in neck blast when the speed of wind was varied. This is related to total number of each organs in a panicle and spots of wound increase proportionally since the number of spikelets and panicle branches is large compared with nodes at the neck of panicles. Therefore, the frequency of wound infection becomes high and panicle branch blast increases.

From these experiments, we have observed that panicle branch blast increases due to wounds caused by wind and the death of panicle branch increases. Since the desease caused by wind was mostly II type rather than IV type or III type, the main effects of wind seem to be infection of spikelet and panicle branch.

V. Infection Period

Examples of panicle branch blast occurring at later period are widely observed recently, and Immano (1933) and Agihara (1958) stressed importance of desease occurring at later period. However, this report does not make clear as to whether this desease at later period is due to infection of later period or incubation of earlier infection. Thus, we have determined incubation period experimentally and attempted to obtain infection period from the period of naturally developed desease.

1. Relation between Infection Period and Incubation Period

a. Experimental Method

Huzisaka no. 5 and Norin no. 1 are used. They were

planted on May 7, and transplanted on June 22. One stock (one stock-4 pieces) was planted in 1/5.000 a Wagner pot. Fertilizer was given as shown in section 1 of chapter II. Three inoculations are made as one each on panicle completion period, milk ripening period and starch ripening period. Blast bacilli (P-2 strain) are inoculated by spray method and kept in humid room of 24-28°C for 36 hours. Afterwards, they were placed in glass room adjusted to 24°C and observed the development of desease. The picking dates were predetermined and pots having the picking status are used eliminating those panicles which did not reach the picking status. From other experiments, we have learned that panicle branch blast does not develop after two weeks from the date of inoculation. We have, therefore, investigated up to 15 days after inoculation.

b. Experimental Results

Table 19 shows the incubation period of spikelet (palea, lemma and glume). The number of deseased spots are highest on 9 th day after inoculation, followed by 6 th and 12th day. Desease is rarely developed on 15th day after inoculation. When we determine average incubation period according to the method of Imura (1940), Huzisaka no. 5 has 9.8 days for panicle completion period, 9.1 days for milk ripening period and 7.7 days for starch ripening period. Norin no. 1 has average incubation period of 9.4 days for panicle completion period, 8.8 days for milk ripening period and 8.0 days for starch ripening period.

Table 19

Incubation Period in Spikelet (palea, lemma, glume)

1) 品種	2) 接種時期	3) 穂数	4) 発病菌所数 5) 6日目	6) 9日目	7) 12日目	8) 15日目	9) 計	10) 平均潜伏期間(日)
11) 関坂5号	穂揃期	47	25	180	68	20	293	9.8
	乳熟期	43	21	75	12	6	114	9.1
	糊熟期	42	56	46	7	—	109	7.7
12) 農林1号	穂揃期	66	35	176	82	11	304	9.4
	乳熟期	53	21	41	6	5	73	8.8
	糊熟期	69	72	66	18	—	156	8.0

13) 備考 1) 発病菌所数は供試総穂数上の全数を示す。
2) 穂揃期接種18/VIII, 乳熟期接種23/VIII, 糊熟期接種28/VIII。

1. Kinds
2. Inoculation period
3. Number of panicles
4. Number of deseased spots
5. 6th day
6. 9 th day

7. 12th day
8. 15th day
9. Total
10. Average incubation period (days)
11. Kuzisaka no. 5 Panicle completion period
Milk ripening period
Starch ripening period
12. Norin no. 1 Panicle completion period
Milk ripening period
Starch ripening period
13. Remark 1) The number of deseased spots indicates total number on panicle.
2) Inoculation at panicle completion period 18/VIII, milk ripening period 23/VIII, starch ripening period 28/VIII

Table 20

Incubation Period in Node of Panicle Branch and Node of Rachis

1) 品種	2) 接種時期	3) 穂数	4) 発病所数 5) 6日目	6) 9日目	7) 12日目	8) 15日目	9) 計	10) 平均潜伏期間 (日)
11) 久坂5号	穂揃期	47	3	8	1	8	20	11.1
	乳熟期	43	11	6	2	7	26	9.6
	糊熟期	42	8	15	4	—	27	8.6
12) 農林1号	穂揃期	66	3	20	2	0	25	8.9
	乳熟期	53	3	0	2	4	9	11.3
	糊熟期	69	3	3	0	—	6	7.5

The keys are same as table 19.

Inoculation soon after panicle formation had longer incubation period in both kinds. Inoculation on old panicles had shorter incubation period. Nodifference in incubation period between the kinds was observed.

Table 20 shows incubation period in node of panicle branch and rachis. According to the table, number of deseases was at the height on 9th day after inoculation and had almost the same tendency as spikelet. Average incubation period of Kuzisaka no. 5 was 11.1 days for panicle completion period, 9.6 days for milk ripening period and 8.6 days for starch ripening period. Norin

:o. 1 had 8.9 days for panicle completion period, 11.3 days for milk ripening period and 7.5 days for starch ripening period. These results do not necessarily agrees with those of Kuzisak no. 5.

Table 21 shows the disease of node at the neck of panicle (neck blast). Average incubation period is long in panicle completion period for both kinds, and medium in milk ripening period whereas it is short in starch ripening period. This has the same tendency as spikelet.

Table 21

Incubation Period in Node at the neck of Panicle

1) 品種	2) 接種時期	3) 穂数	4) 各病斑所数 5)	6)	7)	8)	9) 計	10) 平均潜伏期間 (日)
11) 農林5号	初穂期	47	0	12	1	13	26	12.1
	乳熟期	43	1	0	6	0	7	11.1
	糊熟期	42	0	7	2	—	9	9.7
12) 農林1号	初穂期	66	2	25	6	3	36	9.8
	乳熟期	53	4	5	4	1	14	9.4
	糊熟期	69	3	6	2	—	13	8.3

The keys are same as table 19.

Infection of panicle branch blast can easily occur at relatively later period. Incubation period is somewhat dependent on inoculation time. Our results show tendency of shortening the incubation period as the inoculation was done at later period. Also, incubation period is long for node at the neck of panicle and short for spikelet. Incubation period for node of rachis and panicle branch is in between the above two.

2. Infection Period in Nature

Panicle branch blast is prevented by changing the period of medicinal spraying or combining the period and number of spraying at farm and infection period is investigated from development of panicle branch blast.

a. Experimental Method

Place; Farm of Okayama State Agricultural Experimental Station, Hokuko Okayamashi

Year; 1958-1960 3 years
Kinds; Asahi
Plowing; Planted at middle of may. Transplanted at June [illegible]. 40 stocks are planted per 3.3 square meters. Fertilizer was varied anually and following amount was given (per 10 a). In 1958, ammonium sulfates are given four times as follows. 7.5 Kg on July 31, 7.5 Kg on August 18, 15 Kg on Sept. 1 and 22.5 Kg on Sept. 13. In 1959 and 1960, fertilizer is given on Sept. 5 and July 25 respectively instead of giving at several times.

1) 肥料名	1958年	1959年	1960年
2) 硫安アンモニア (元肥)	37.5kg	80kg	150kg
3) 同上 (追肥)	22.5	12	27
4) 過リン酸石灰	15	40	40
5) 塩化カリウム	15	20	20
6) 大豆粕	30	80	80
7) 堆肥	1,500	1,200	1,000

1. Fertilizer
2. Ammonium sulfate (original)
3. Same as above (follow)
4. Calcium phosphate
5. Potassium salt
6. Soy bean
7. Compost

Table 22

Weather table 1 (1958) (Examined by weather observatory of Okayama Region)

1) 月	2) 半旬	3) 最高気温 °C	4) 最低気温 °C	5) 平均気温 °C	6) 降水量 mm	7) 降水日数 日	8) 日照時間 時間	14) 備考
9) 6 月	1	27.0	14.1	20.4	7.7	1	44.7	
	2	24.6	15.5	20.1	25.3	4	21.4	
	3	27.3	17.0	21.7	29.9	2	47.8	
	4	28.6	15.6	21.7	0.9	2	51.2	
	5	29.0	17.8	23.1	4.9	1	42.7	
	6	28.7	22.3	25.2	101.7	5	15.9	15) 雨呉 (29日)
10) 7 月	1	28.1	22.6	24.8	59.8	5	8.7	16) つみあげ (10日)
	2	30.0	22.9	26.2	6.5	3	27.8	
	3	31.3	22.4	26.3	0.3	1	45.5	
	4	31.5	23.2	27.1	1.8	3	43.4	
	5	32.9	24.2	27.8	1.2	4	31.6	17)
	6	33.6	24.0	27.8	1.0	2	48.4	追肥 (31日)
11) 8 月	1	33.4	24.5	27.6	5.9	3	36.6	
	2	31.5	21.1	26.0	6.1	1	30.5	
	3	31.1	22.9	26.0	31.4	4	28.2	18)
	4	30.9	22.3	26.1	1.0	2	31.0	追肥 (18日)
	5	28.0	21.7	24.8	83.0	5	12.4	台風 (23, 24日)
	6	30.6	23.1	26.9	13.9	4	35.0	19)
								20)
12) 9 月	1	29.7	21.1	24.7	4.2	2	27.4	追肥(1日)薬剤散布(6日)
	2	31.2	22.0	25.9	3.0	3	32.8	出穂期 (10日)
	3	29.8	20.9	24.9	26.7	4	33.5	追肥 (13日)
	4	28.8	19.5	23.1	0.0	2	34.2	薬剤散布 (11, 15日)
	5	25.2	18.5	21.1	49.5	4	11.8	台風 (17日, 雨なし)
	6	25.0	15.9	19.9	1.3	3	31.3	大雨 (22, 23日)
								薬剤散布 (24日)
								薬剤散布 (30日)
13) 10 月	1	21.8	13.3	18.3	17.2	2	17.6	21)
	2	22.7	9.4	15.1	1.4	2	39.7	薬剤散布 (8日)
	3	24.9	15.4	19.7	22.5	2	18.7	
	4	20.4	12.1	15.8	35.2	3	17.6	雨 (17, 18日)
	5	20.5	11.3	15.4	9.8	4	26.2	
	6	17.3	6.8	11.5	6.6	2	25.8	

1. Month
2. Period of 5 days
3. Highest temperature
4. Lowest temperature
5. Average temperature
6. Amount of rain fall
7. Number of days of rain fall
8. Time of sun shine
9. June
10. July
11. August
12. September
13. October
14. Remarks
15. Farm plant (29th day)
16. Tsuyuake (10th day)
17. Additional fertilizer (31st day)
18. Additional fertilizer (18th day)
19. Strong wind (23rd, 24th day)

20. Additional fertilizer (1st day) medicine spray (4th day)
Panicle formation period (10th day). Additional fertilizer
(13th day) Medicine spraying (14th, 15th day)
Strong wind (17th day. no rain)
Big rain (22nd, 23rd day)
Medicine spraying (24th day)
Medicine spraying (30th day)
21. Medicine spraying (9th day)
Rain (17th, 18th day)

Table 23

Weather Table 2 (1959) (Examined by weather observatory of Okayama Region)

1)	2)		3)	4)	5)	6)	7)	8)	14)
	月		気温	気温	気温	降水量	日照時間	風速	備考
			℃	℃	℃	mm	h.	km/h	
9)	6 月	1	26.[illegible]	[illegible].0	20.2	5.6	3	39.1	
		2	25.9	15.9	21.9	67.1	4	33.8	
		3	25.9	14.5	19.9	0.0	0	44.1	
		4	28.8	18.8	23.3	1.9	2	31.8	
		5	28.8	19.1	23.5	0.3	3	32.8	
		6	28.9	19.7	24.1	10.5	3	38.9	
10)	[illegible] 月	1	30.3	22.8	26.1	11.2	4	24.2	
		2	30.4	22.7	26.3	18.1	5	26.9	15)
		3	28.2	22.6	25.0	80.3	5	19.6	雨雨 (11, 13~15日)
		4	30.3	23.6	26.1	6.4	3	20.1	
		5	32.0	23.8	27.5	0.1	1	33.9	
		6	33.3	21.5	26.9	0.0	2	63.8	
11)	8 月	1	34.8	22.5	28.4	0.0	0	59.6	16)
		2	30.0	24.2	26.4	43.1	4	24.3	台風 (8, 9日)
		3	29.1	21.8	24.4	5.0	1	28.7	
		4	31.5	23.3	26.9	9.0	1	31.2	
		5	31.7	23.2	26.8	0.2	4	32.7	農薬散布 (25日)
		6	33.8	23.1	27.8	0.6	0	60.2	農薬散布 (28日)
12)	9 月	1	32.0	21.0	25.9	0.8	1	38.2	[illegible]日) 17)
		2	30.7	20.4	25.1	1.7	3	31.4	農薬散布(5日) [illegible]剤散布 (10日)
		3	30.0	21.7	25.1	65.7	4	17.1	農薬散布 (15日)
		4	28.3	21.0	24.8	10.1	2	37.5	台風 (17, 18日)
		5	27.2	17.1	21.9	45.3	3	27.0	農薬散布 (23日)
		6	25.6	16.0	20.4	65.6	2	33.1	台風 (26日)
13)	10 月	1	23.1	16.9	19.5	12.5	3	12.1	
		2	24.8	14.4	18.7	35.3	2	28.2	
		3	22.6	9.5	15.4	0.0	0	38.6	
		4	21.0	11.6	15.7	25.0	2	23.1	
		5	23.9	9.7	15.8	0.0	0	40.9	
		6	22.4	11.7	16.5	6.2	3	40.1	

Keys are same as table 22 except following.

15. Heavy rain (11th, 13-15th day)
16. Strong wind (?th, ?th day)
    Medicine spraying (25th day)
    Medicine spraying (?7th day)
17. Additional fertilizer (5th day)
    Medicine spraying (?th day)
    Panicle formation period (10th day)
    Medicine spraying (15th day)
    Strong wind (17th, 18th day)
    Medicine spraying (23rd day)
    Strong wind (24th day)

Medicine spraying:
Spraying period:
Number of spraying:
Spraying concentration;
} These are different for each experiment and the results are shown in tables.

Amount of spraying: 4 Kg powder per 10 a and 130 l liquid per 10a.
Classification; 3-4 continuous sections lump method.
Area; One section: 15-16.5 square meters

Table 24

Weather Table 3 (1960) (Examined by weather observatory of Okayama Region)

Keys are same as table 22 except following

15. Big rain (21st, 22nd day)
16. Big rain (?th day)
    Dawn high temperature
17. Strong wind (12th day)
    Strong wind (29th day)
18. Medicine spraying (1st day)
    Medicine spraying (2?th day)
    Panicle formation period (11th day)
    Medicine spraying (21st day)
    Medicine spraying (?8th day)
19. Big rain (6th, 7th day)

Examination; In middle-end part of October, deseases are examined on 20-30 stocks in one section. Node blast and neck blast are compared by panicle desease ratio. Panicle branch blast is based on index value of chapter II, and panicle desease is examined in 10 separate classifications. Following equation was used for panicle branch desease.

$$\text{Panicle branch desease} = \frac{\Sigma(\text{Desease of each panicle})}{\text{Total number of panicle}} \times 100\ (\%)$$

Panicle desease rate is licht for degree of desease 0.1-0.3, medium for 0.4-0.7 and heavy for 0.8-1.0.

Weather; Weather of June-October in 5 days period was shown in table 22-24.

b. Experimental Results.

(1) Investigation by Seresan lime spraying

This experiments are conducted in 1956-1958. In 1958, experiments on number of spravings are conducted and the results are shown in table 25.

1) 月	2) 半旬	3) 最高気温 °C	4) 最低気温 °C	5) 平均気温 °C	6) 降水量 mm	7) 雨天日数 日	8) 日照時間 時間	14) 備考
9) 6 月	1	24.5	15.0	19.6	11.6	5	26.1	
	2	29.5	14.7	21.8	1.2	1	53.7	
	3	22.4	16.2	18.7	24.0	5	12.6	
	4	26.4	16.3	20.8	4.1	3	27.0	
	5	26.5	18.8	22.0	86.4	4	19.7	15) 大雨 (21, 22日)
	6	30.6	22.0	26.0	0.0	1	31.7	
10) 7 月	1	31.0	22.5	25.7	23.5	3	30.1	16)
	2	29.0	22.0	25.1	128.1	3	21.7	大雨 (8日)
	3	32.1	21.8	26.6	4.2	2	54.3	つゆ明け高温
	4	32.7	22.7	26.2	0.8	3	34.7	
	5	33.2	23.3	27.8	0.0	0	51.9	
	6	33.0	23.9	27.8	31.2	3	52.3	
11) 8 月	1	34.4	24.4	28.9	0.0	1	50.4	
	2	33.2	23.7	28.0	20.9	1	54.0	17)
	3	29.2	23.2	25.1	121.1	4	19.7	台風 (12日)
	4	31.5	22.5	26.4	0.0	0	44.6	
	5	32.4	23.4	27.4	0.3	2	42.0	
	6	31.5	22.5	26.6	45.5	3	40.4	台風 (29日)
12) 9 月	1	28.8	22.3	24.8	40.2	5	7.5	薬剤散布 (1日) 18)
	2	27.5	19.3	22.1	23.6	3	19.2	薬剤散布 (8日)
	3	27.0	20.9	22.1	19.5	4	3.7	出穂期(11日)出穂不揃
	4	29.6	20.6	24.1	32.2	2	23.1	
	5	28.8	18.2	22.6	0.6	2	29.6	薬剤散布 (21日)
	6	25.4	15.9	20.0	4.3	5	23.9	薬剤散布 (28日)
13) 10 月	1	22.6	13.9	17.7	4.3	3	15.5	19)
	2	22.7	14.6	18.1	72.1	2	20.1	大雨 (6, 7日)
	3	25.2	12.0	18.1	0.0	0	41.5	
	4	23.3	11.3	16.4	20.9	2	36.1	
	5	22.6	6.3	14.2	0.0	0	42.5	
	6	19.7	7.9	13.1	11.1	2	35.1	

Table 25

Experiment by Spraying Seresan line (1) (1952) (Average of 3 sections)

1) 散布区	2) 節イモチ 罹病率 %	3) 節イモチ 穂首穂枯率 %	4) 枝梗イモチ 罹病率 %	4) 枝梗イモチ 穂枯率 %	5) 軽罹病率 %	6) 中罹病率 %	7) 重罹病率 %	8) 枝梗被害度 %
9) 穂孕期 1回散布	0.2(96)	0.4(73)	4.1(61)	2.7(10)	1.2(0*)	3.2(22)		
10) 孕穂・穂揃期 2回散布	0.6(89)	0.3(80)	3.1(70)	2.0(33)	1.7(0*)	3.1(21)		
11) 孕穂～乳熟期 3回散布	0.3(94)	0.4(73)	5.0(52)	2.8(7)	1.0(0*)	3.3(20)		
12) 孕穂～糊熟期 4回散布	0.3(94)	0.1(93)	2.6(75)	1.1(63)	0.3(63)	1.2(71)		
13) 孕穂～黄熟初期 5回散布	0.0(100)	0.1(93)	2.2(79)	1.0(67)	0.6(25)	1.4(66)		
14) 標準無散布	5.3(—)	1.5(—)	10.5(—)	3.0(—)	0.8(—)	4.1(—)		

15) 備考 1) 散布月日は次のとおりである。孕穂期散布 6/IX, 穂揃期散布 15/IX, 乳熟期散布 24/IX, 糊熟期散布 30/IX, 黄熟初期散布 8/X。

2) カッコ内は防除率を示す。

$$\text{防除率} = \frac{(\text{無散布区発病率}) - (\text{散布区発病率})}{\text{無散布区発病率}} \times 100(\%)$$

3) *印：計算上は負数になるが実際上防除効果がなかったことになるから 0 として表示した。以下これに準ずる。

1. Spraying
2. Node blast ratio
3. Neck blast of panicle desease ratio
4. Panicle branch blast
5. Light panicle desease ratio
6. Medium panicle desease ratio
7. Heavy panicle desease ratio
8. Degree of panicle branch desease
9. Panicle formation period, one spray
10. Panicle formation; completion period: two sprays
11. Panicle formation-Milk ripening period, three sprays
12. Panicle formation-Starch ripening period, four sprays
13. Panicle formation-Initial yellow ripening period, five sprays
14. Standard, no spray
15. Remark 1) Spray dates are as follows. Panicle formation period 6/IX, panicle completion period 15/IX, milk ripening period 24/IX, starch ripening period 30/IX, initial yellow ripening period 8/X.

2) Parenthesis indicates prevention ratio

$$\text{Prevention ratio} = \frac{(\text{Desease ratio in no spray section}) - (\text{Desease ratio in spray section})}{\text{Desease ratio of no spray section}} \times 100(\%)$$

3) Seal; Negative numbers from calculation are treated as zero prevention effect. This is followed from here on.

According to table 25, desease ratio of light panicle branch blast was low in spray section compared with no spray section. Between each spray sections, the difference in light panicle desease ratio was not large, and the ratio was a little low in 4-5 times spray sections. Panicle desease ratio of medium and heavy symptoms was high in 1-3 times spray section from panicle formation period to milk ripening period and no difference was observed in standard, no spray section. However, spraying at starch ripening period in addition to three times of spray reduced the panicle desease ratio. Panicle desease ratio for five sprays in initial yellow ripening period was about same as that for four sprays. Degree of panicle branch desease was high in 1-3 sprays section and low in 4-5 sprays section. Therefore, spray in starch ripening period reduced the desease.

Looking back at weather conditions since 1953 (see table 22), there were strong wind without rain on Sept. 17 and big rain on 22nd and 23rd right before the spray of milk ripening period (24/IX). Weather afterwards was relatively smooth.

Considering all these conditions effects of medicine spray were reduced due to wind and rain of middle-end of September, and infection was rapidly spread after the strong wind. Thus, the opportunity for infection was increased from the end of September to beginning of October. Thus, spraying on Sept. 30 of starch ripening period prevented bacilli invasion and considered to reduce desease.

Table 26

Experiment by Spreading Seresan Line (2) (1959)
(Average of 3 sections)

1) 散布区	2) 節イモチ	3) 節イモチ	4) 穂首イモチ			
			5) 軽症	6) 中症	7) 重症	8) 指数
	%	%	%	%	%	%
9) 幼穂形成中期 1回散布	5.5(84)	5.5(—)	10.4(46)	3.8(72)	3.1(77)	6.7(71)
10) 幼穂形成後期 1回散布	6.9(80)	5.8(76)	16.1(16)	8.1(40)	3.4(75)	10.1(56)
11) 穂孕期 1回散布	6.2(82)	6.7(73)	15.0(22)	7.4(46)	5.9(57)	11.8(49)
12) 穂揃期 1回散布	10.4(70)	10.6(57)	18.6(3)	8.2(40)	6.2(54)	13.4(42)
13) 乳熟期 1回散布	6.2(82)	16.1(30)	19.6(0)	5.5(59)	5.9(57)	11.8(49)
14) 幼穂形成中期・穂揃期 2回散布	2.1(94)	3.2(87)	4.6(76)	1.4(90)	2.3(83)	3.7(84)
15) 幼穂形成後期・穂孕期 2回散布	2.2(94)	1.9(92)	9.1(52)	2.0(85)	1.1(92)	3.7(84)
16) 穂孕期・穂揃期 2回散布	4.7(86)	3.3(87)	14.3(25)	3.6(74)	2.3(83)	6.4(72)
17) 穂揃期・乳熟期 2回散布	3.5(90)	5.3(78)	16.3(15)	4.3(68)	2.0(85)	6.7(71)
18) 慣行無散布	34.2(—)	24.4(—)	19.1(—)	13.6(—)	13.6(—)	23.2(—)
F_0	**	**	n.s.	**	**	**
t (0.01) Sd	5.86	11.48	—	3.16	4.02	4.49
t (0.05) Sd	4.28	8.38	—	2.31	2.94	3.27

19) 備考 1) 散布月日は次のとおり。幼穂形成中期散布 25/Ⅷ, 幼穂形成後期散布 28/Ⅷ, 穂孕期散布 5/Ⅸ, 穂揃期散布 15/Ⅸ, 乳熟期散布 23/Ⅸ。
2) カッコ内は防除率を示す。

1. Spreading
2. Node blast desease ratio
3. Neck blast desease ratio
4. Panicle branch blast
5. Light panicle desease ratio
6. Medium panicle desease ratio
7. Heavy panicle desease ratio
8. Degree of panicle branch desease
9. Middle period of young panicle formation, one spreading
10. Latter period of young panicle formation, one spreading
11. Panicle completion period, one spreading
12. Panicle completion period, one spreading
13. Milk ripening period, on spreading
14. Middle period of young panicle formation; Panicle completion period. Two spreading
15. Latter period of young panicle formation. Panicle formation period. Two spreading
16. Panicle formation period. Panicle completion period Two spreading
17. Panicle completion period. Milk ripening period Two spreading

18. Standard, No spreading
19. Remark 1) Spreading dates are as follows. Spreading at middle period of young panicle formation 25/VIII, latter period of young panicle formation 29/VIII, Panicle formation period 5/IX, Panicle completion period 15/IX, Milk ripening period 23/IX
2) Parenthesis indicates prevention ratio

Table 26 shows experimental results obtained in 1959 by spreading period of Seresan line. According to table 26, desease is small in one spreading at middle period of young panicle formation (25/VIII) but many deseases are developed at other periods. However, the desease is reduced considerably by two spreadings as a result of combining two periods.

Specifically, panicle desease is the least in two spreading during middle period of young panicle formation and panicle completion period (25/VIII, 15/IX) and is less in two spreadings during latter period of young panicle formation and panicle formation period (29/VIII, 5/IX) and one spreading during middle period of young panicle formation (25/VIII). Panicle deseases in other cases were many in both one or two spreadings. Medium and serious panicle deseases were small in one spreading at middle period-latter period of young panicle formation and many in other cases. Two spreadings reduced panicle deseases specially during middle period of panicle formation, panicle completion period and latter period of panicle formation. Panicle branch decease has a tendency of medium and serious panicle desease ratio.

Weather of 1959 (see table 23) indicates that the clear weather was disturbed by strong wind of August 8-9 and rainy days continued until August 15. Temperature was little lower than usual. After august 15, the weather was good with small amount of rain and high temperature extended to beginning of September. From second 5 days of September, there was rain and strong wind blew on Sept. 17 and 26. Weather was bad between these two winds.

Blast desease began to be active under unseasonable weather in the first half of August. But the second infection was checked by medicine spreading (25/VIII) during middle period of young panicle formation. The spread of blast desease after the middle period of young panicle formation was small because of good weather in second half of August. Thus, the frequency of infection was considerably reduced and the development of desease was very small under these conditions.

whereas node blast and neck blast became relatively small by one spreading, panicle branch blast desease was not much reduced. The reason is probably as follows. Due to the effects of strong wind (Sept. 17, Sept. 26), environment became better for development of desease and opportunity for blast bacilli invasion existed at the end of September and beginning of October same as in 1958. However, in node and neck blast, the deseased was few in spite of bacilli invasion during this period. On the other hand, the panicle death increased as a result of infection during this period.

(2) Investigation by Spreading PMA Emulsion

Experimental results obtained by using PMA emulsion in 1959 are shown in table 27.

Table 27

Experiment by Spreading PMA Emulsion (1958) (Average of 3 sections)

	1) 散布区	2) 節イモチ 罹病率	3) 首イモチ 病穂率	4) 枝梗イモチ 5) 軽 病穂率	軽 病穂率	6) 中 病穂率	中 病穂率	7) 重 病穂率	重 病穂率
		%	%	%		%		%	
8)	穂孕期 1回散布	0.1	0.0	5.4(14)		1.3(54)		6.7(26)	
9)	出穂期 1回散布	0.4	0.0	5.7(10)		1.5(46)		7.2(21)	
10)	穂揃期 1回散布	0.3	0.2	4.7(25)		1.9(32)		6.6(28)	
11)	乳熟期 1回散布	0.4	0.1	6.9(5)		3.2(0)		9.2(0)	
12)	穂孕・出穂期 2回散布	0.1	0.1	5.0(21)		2.7(4)		7.7(15)	
13)	穂孕・穂揃期 2回散布	0.0	0.0	3.6(43)		1.1(61)		4.7(48)	
14)	穂揃・乳熟期 2回散布	0.0	0.4	4.8(24)		3.9(0)		8.7(4)	
15)	標準無散布	0.4	0.2	6.3(—)		2.8(—)		9.1(—)	

16) 備考 1) 散布月日は，穂孕期散布 6/IX，出穂期散布 11/IX，穂揃期散布 15/IX，乳熟期散布24/IX。
2) カッコ内は防除率を示す。
3) PMA乳剤は1958年7月に農林省から分譲を受けたもので水銀含量は Hg として 4.5%，散布濃度は2,000倍。

1. Spreading
2. Node blast ratio
3. Panicle desease ratio of neck blast
4. Panicle branch blast
5. Rate of light panicle desease
6. Rate of medium panicle desease
7. Rate ofserious panicle desease
8. Panicle formation period, one spreading
9. Panicle growth period, one spreading

10. Panicle completion period, one spreading
11. Milk ripening period, one spreading
12. Panicle formation, panicle growth period, two spreading
13. Panicle formation, panicle completion, Two spreading
14. Panicle completion, milk ripening period, two spreading
15. Standard, no spreading
16. Remark 1) Spreading dates; panicle formation period 6/IX panicle growth period 11/IX, panicle completion period 15/IX, milk ripening period 24/IX.
2) Parenthesis indicates prevention ratio.
3) PMA emulsion was obtained in July, 1958 from Agricultural Technology Research Institute, and contains 4.5 % Hg. Spreading concentration: 2,000 times.

Deseases at this farm were generally less. Deseases were developed a little more in section of one spreading. No large difference between the spreading sections was observed and the deseases were developed less in section of two spreadings at panicle formation and panicle completion period. Deseases were less for spreading at panicle formation period (6/IX) or panicle completion period (15/IX) and a little more for spreading at milk ripening period (24/IX). Deseases were in between above two for spreading at panicle growth period. Deseases were probably developed a little more for spreading during milk ripening period due to washing of medicine by bad weather. Panicle branch blast was less developed through 1-2 spreading during panicle formation period because medicine lowered conidial density of blast bacilli and reduced infection of young panicle. Also the spreading during panicle completion period was useful in preventing infections. In 1958, the most infectious period of panicle branch blast was considered to be the end of September as mentioned previously. In this experiment, it was difficult to determine infectious period because the medicinal spreading was not conducted during this period.

(3) Investigations by Spreading Blast-Siding S

Table 23 shows experimental results from spreading 30 ppm blast siding S in 1960. According to table 28, development of light desease in panicle branch blast was a little more in section of one spreading. There was almost no difference in development of desease between the spreading section of both panicle formation period (1/IX) and starch ripening period (23/IX) and no spreading section. Panicle desease rate in two spreading section including panicle formation period was slightly lower. In medium and serious deseases, panicle desease rate was slightly lower in one spreading section but no difference existed between the spreading periods.

Table 2*

Experiment of Spreading Blast-Siding S (1970)
(Average of 4 sections)

	1) 散布区		2) 穂イモチ病穂率	3) 枝梗イモチ			
				4) 罹病率	5) 中発生	6) 多発生	7) 被害度
8)	穂孕前期(プラS)	1回散布	10.7(46)	10.9(10)	2.7(57)	2.6(45)	5.6(43)
9)	穂孕後期(プラS)	1回散布	4.3(78)	7.5(38)	2.2(65)	2.5(47)	4.8(52)
10)	乳熟期(プラS)	1回散布	11.6(42)	9.2(21)	2.8(56)	2.2(53)	5.1(48)
11)	糊熟期(プラS)	1回散布	10.4(48)	11.6(4)	2.7(57)	2.0(57)	4.9(51)
12)	穂孕前期(プラS)・同上(プラS)	2回散布	3.2(84)	7.7(36)	1.4(78)	1.5(68)	3.2(69)
13)	穂孕前期(水 銀)・同上(水 銀)	2回散布	3.3(83)	5.9(51)	0.7(89)	1.4(70)	2.5(75)
14)	穂孕前期(プラS)・同上(水 銀)	2回散布	4.8(76)	6.6(45)	1.7(73)	1.8(62)	3.3(67)
15)	穂孕前期(水 銀)・同上(プラS)	2回散布	4.5(77)	6.3(48)	2.0(68)	1.6(66)	3.5(65)
16)	穂孕後期(プラS)・乳熟期(プラS)	2回散布	2.7(86)	5.3(56)	0.7(89)	0.4(91)	1.8(82)
17)	穂孕後期(水 銀)・同上(水 銀)	2回散布	4.9(75)	5.9(51)	1.3(79)	1.8(62)	3.1(69)
18)	穂孕後期(プラS)・同上(水 銀)	2回散布	4.7(76)	7.4(39)	1.0(84)	1.5(68)	3.0(70)
19)	同上(水 銀)・同上(プラS)	2回散布	1.8(91)	3.7(69)	0.3(95)	0.8(83)	1.3(87)
20)	無 散 布		19.9( )	12.1(—)	6.3(—)	4.7(—)	9.9(—)
	F_0		**	**	**	n.s.	**
	t (0.01) Sd		9.67	4.73	3.23	—	4.69
	t (0.05) Sd		7.21	3.52	2.41	—	3.49

21) 備考 1) 散布月日は，穂孕前期散布 1/IX，穂孕後期散布 8/IX，乳熟期散布 21/IX，糊熟期散布 28/IX。
2) プラスサイジンSは 30 ppm，水銀はフミロンの実用濃度（水 15 l にフミロン錠 2 錠の割合）とした。
3) カッコ内は調査率を示す。

1. Spreading section
2. Panicle desease rate of neck blast
3. Panicle branch blast
4. Panicle desease rate in light desease
5. Panicle desease rate in medium desease
6. Panicle desease rate in serious desease
7. Degree of panicle branch desease

8. First half period of panicle formation (Bla S), one spreading
9. Latter half period of panicle formation(Bla S), one spreading
10. Milk ripe period (Bla S), one spreading
11. Starch ripe period (Bla S), one spreading
12. First half panicle formation period (Bla S), Latter half period (Bla S), two spreadings.
13. Same as above (mercury), same as above (mercury), two spreadings
14. Same as above (Bla S), same as above (mercury), two spreadings
15. Same as above (mercury), same as above (Bla S), two spreadings
16. Latter half of panicle formation period (Bla S), milk ripe period (Bla S), two spreadings
17. Same as above (mercury), same as above (mercury), two spreadings
18. Same as above (Bla S), same as above (mercury) two spreadings
19. Same as above (mercury), same as above (Bla S). two spreadings
20. Standard, no spreading
21. Remark 1) Spreading dates, First half of panicle formation period 1/IX, latter half of panicle formation 8/IX, milk ripe period 21/IX, starch ripe period 28/IX.
    2) 30 ppm blast siding S and mercury was the actual fumiron concentration. (two fumiron tablets per 15 liters of water)
    3) Parenthesis indicates prevention rate

While panicle desease rate for each period was not much different in serious and medium desease, the desease rate for starch ripe period was high in light desease. These are considered to be due to the infections after starch ripe period. Also, a few deseases in two spreading sections including panicle formation period were due to the reduction of infectious sources. In two spreadings of blast siding S or this and mercury, panicle branch blast was specially few in milk ripe period. This indicates a large effect of blast siding S spreading during milk ripe period.

Considering the experimental results for three years, panicle branch blast was checked by the spreading during starch ripe period in 1958 (table 25), and the desease was specially reduced by spreading during panicle growth period in addition to young panicle formation period in 1959 (table 26). In 1960, the results are almost similar to previous year (table 27). The reason for the decrease of deseases by medicinal spreading before panicle growth period is that the spreading in this period reduces density of infectious source and decrease infections after panicle growth. The reason for the decrease of

desease by medicinal spreading after panicle growth period is that it mainly prevents the infections. From these results, we can say that panicle branch blast is infected gradually over long period of time from panicle formation to starch ripe period. The most infectious period was strongly affected by weather and different from year to year.

3. Summary

Inoculation period of panicle blast branch is different depending on the parts of initial infection and infectious period, but was about 7.5-11 days. Medicinal spreadings during each period from panicle formation to beginning of yellow ripe period were effective in some cases and ineffective in other cases. Considerations of conditions during medicinal spreading and incubation period indicate that panicle branch blast is infected over the long period of time from panicle formation to starch ripe period.

Infections of spikelet and panicle branch by inoculation are clearly shown by experimental observations of chapter II and even clearer in chapter VI. Of course, the most infectious period differs considerably from year to year due to variable yearly environment, but it is clear that panicle branch blast is easily occurred by infection during later period.

VI Infection at Later Period and Damage

Although infections of panicle branch blast are known to occur over the long period of time, damages due to infections at later period are not clear. Thus, we have attempted to verify these from outdoor experiment.

1. Experimental Method

Aichiasahi (weak resistance) and Tosan no. 38 (strong resistance) were studied outdoor in a pot (inner square 50 x 50 cm, depth 30 cm and concrete bottom) about 5 cm of gravel is packed at the bottom of pot, and about 18 cm of field soil is placed on top of it. As fertilizers, 40 g of ammonia sulfate, 60 g of phosphate lime and 16 g of potassium sulfate are given. Planted on May 7. and transplanted on June 22. 9 stocks (4 pieces per one stock) were planted in a pot. Additional 5 g of ammonium sulfate were given on July 20 and August 21. Three inoculation periods of panicle completion period, milk ripe period and starch ripe period (for Tosan no. 38, yellow ripe period was added) were established and two pots of each kind are used for one period. Three stocks per pot, total 6 stocks were used without inoculation.

Rice plant without inoculation was covered with polyethylene bag and isolated from adhesion of conidium. Inoculation of bacilli (P-2 strain) was conducted by spray method and panicles were covered with polyethylene bag immediately afterwards. They are left two days. Each plants are marked by the dates so that inoculation dates can be traced. Number of infections was examined 13 days after inoculation, and degree of panicle branch desease was examined 3 weeks after inoculation and October 24-25. Examination of desease was conducted by the method of chapter II.

Experiment of later period infection was conducted with panicles having same date of forming node at the neck of panicle. Thus, the size of panicle was made even. In experiment of Aichiasahi, node at the neck of panicle was formed at 9-10th of September and in Tosan no. 3 , it was formed on 5-6 th of September. Desease of panicle branch whose node at the neck was not invaded was examined on 3 weeks after inoculation. The panicles are cut at the end of October and dried by wind for about a month. Panicle weight, number ratio and weight of 1,000 grains unpolished rice were measured.

These experiments are conducted at Agricultural Technology Research Institute (Nishikehara Kitaku Tokyo) in 1956 and the weather of that year is shown in Figure 6.

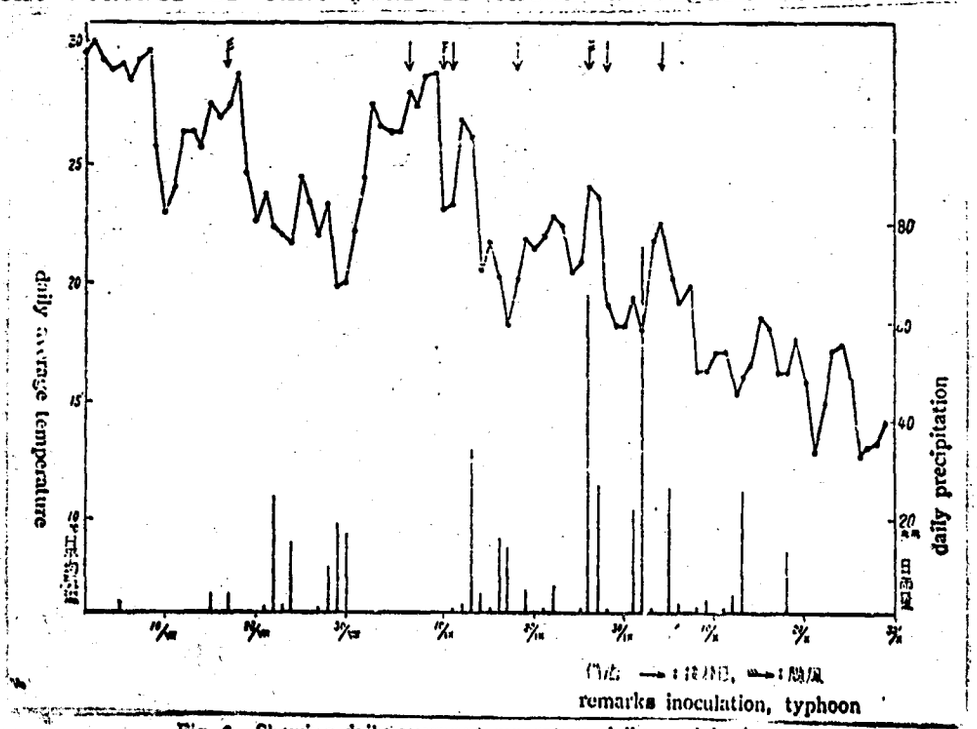

Fig. 6.—Showing daily average temperature, daily precipitation, date of typhoon and date of inoculation during the course of the outdoor experiment in 1956.

2. Experimental Results

a. Infection at Later Period and Develcpment of Desease

Distribution of panicles during inoculation by number of days after node at the neck of panicle is formed is shown in table 29.

Table 29

Distribution of Panicles during Inoculation by Number of Days after Node at the Neck of Panicle is formed

1) 穂首抽出後日数 (日)	2) 愛知旭 A区 (本)	愛知旭 B区 (本)	愛知旭 C区 (本)	3) 東山38号 A区 (本)	東山38号 B区 (本)	東山38号 C区 (本)
−3	33			36		
−2						
−1						
0	37			36		
1	22			40	7	
2	26			37	22	
3	14			9	31	
4	8				38	
5	13				40	
6	4	24			29	
7	2	32			7	
8		23			1	
9		37				10
10		9				14
11		8				40
12		13				19
13		1				12
14			15			6
15						1
16			21			
17			14			
18			22			
19			32			
20			14			
21			12			
22			13			
23			6			
24			1			
計	159	147	150	158	175	102

4) 備考 1) 愛知旭A区は11/IX接種, B区は18/IX接種, C区は 28/IX接種
2) 東山38号区は6/IX 接種, B区は11/IX日接種, C区は18/IX日接種

1. Number of days after node at the neck of panicle is formed
2. Aichiasahi, A section, B section, C section
3. Tosan no. 38. A section, B section, C section
4. Remark 1) A section of Aichiasahi was inoculated at 11/IX, B section 18/IX and C section 28/IX
   2) A section of Tosan no. 38 was inoculated at 6/IX, B section 11/IX and C section 18/IX.

According to table 29, Aichiasahi was examined from 3 days before to 24 days after node at the neck of panicle was formed. Panicles used during panicle completion period are from 3 days before to 7 days after node at the neck of panicle was formed when considered by classification of inoculations and most of them are the panicles from immediately after to 3 days after the node formation. In inoculation during milk ripe period, the panicles are 6-13 days after the node formation and most of them are 6-9 days after neck formation. In inoculation during starch ripe period, the panicles are 13-24 days after the node at the neck of panicles was formed, and most of them are 16-20 days. Similarly in Tosan no. 38, panicles for completion period are from immediately after to two days after node at the neck of panicle was formed, 2-6 days after node formation for milk ripe period and 10-13 days after node formation for starch ripe period. Although the above examinations are lacking during yellow ripe period, most of them are estimated to be 25-29 days after node at the neck of panicle was formed since panicle growth period was Sept. 3 and inoculation was conducted on Oct. 4. The period when more than half had formed node at the neck of panicles corresponds to 3-4 days after the growth period.

Table 30 shows number of infections on each parts of panicle on 13th day after inoculation.

According to table 30, many deseases were developed in spikelet and particularly severe for inoculation at later period. Tosan no. 38 had 9 spots of deseases per panicle for inoculation at starch ripe period. Deseases on node of panicle branch or rachis were very few compared with spikelet. However, the number of infections increases for inoculation at latter period similar to the case of spikelet.

Table 39

Inoculation at Later Period and Infections

1) 品種	2) 接種月日	3) 穂数	4) 1穂当たり感染数 5) 内外穎	6) 護穎	7) 内外護穎	8) 穂軸節	9) 枝梗節	10) 退化枝梗	11) 穂首節
12) 愛知旭	9.11 (乳熟期)	159	1.1	0.03	2.0	0.1	0.2	0.1	0.32
	9.18 (乳熟期)	147	0.4	0.2	3.0	0.4	0.4	0.1	0.34
	9.28 (初糊期)	150	—	—	—	—	1.1	—	0.44
	無接種	148	0	0	0	0	0	0	0
13) 東山38号	9.6 (乳熟期)	158	0.4	0.02	0.8	0.03	0.02	—	0.09
	9.11 (乳熟期)	175	0.3	0.01	1.6	0.02	0.1	—	0.11
	9.18 (糊熟期)	102	0.2	0.1	8.5	0.2	0.7	0.03	0.15
	10.4 (黄熟期)	113	—	—	—	—	--	—	0.10
	無接種	173	0	0	0	0	0	0	0

1. Kinds
2. Inoculation date
3. Number of panicles
4. Number of infections per panicle
5. Palea and lemma
6. Glume
7. Inner and outer lemma
8. Node of panicle branch
9. Node of rachis
10. Devoluted panicle branch
11. Node at the neck of panicle
12. Aichiasahi (panicle completion period
(milk ripe period)
(starch ripe period
(no inoculation
13. Tosan no. 38 (panicle completion period
(milk ripe period)
(starch ripe period)
(yellow ripe period)
no inoculation

Table 31

Inoculation at Later period and Development of Desease

1) 品種	2) 接種月日	3) 接種後3週間目 4) 枝梗イモチ	5) 首イモチ	6) 計	7) 10月24日 8) 枝梗イモチ	9) 首イモチ	10) 計
		%	%	%	%	%	%
11) 愛知旭	9.11 (穂揃期)	18.7 (49.5)	19.1 (50.5)	37.8 (100)	24.1 (35.0)	44.7 (65.0)	68.8 (100)
	9.18 (乳熟期)	28.6 (63.1)	16.7 (36.9)	45.3 (100)	36.0 (50.8)	34.8 (49.2)	70.8 (100)
	9.28 (糊熟期)	61.6 (67.2)	30.0 (32.8)	91.6 (100)	61.6 (67.2)	30.0 (32.8)	91.6 (100)
	無接種	0	0	0	0	0	0
12) 農山38号	9.6 (穂揃期)	6.9 (43.1)	9.1 (56.9)	16.0 (100)	22.8 (51.0)	21.9 (49.0)	44.7 (100)
	9.11 (乳熟期)	13.5 (55.8)	10.7 (44.2)	24.2 (100)	26.2 (40.0)	39.3 (60.0)	65.5 (100)
	9.18 (糊熟期)	37.0 (76.9)	11.1 (23.1)	48.1 (100)	47.8 (57.2)	35.7 (42.8)	83.5 (100)
	10.4 (黄熟期)	72.0 (91.7)	6.5 (8.3)	78.5 (100)	72.5 (91.7)	6.5 (8.3)	78.5 (100)
	無接種	0	0	0	0	0	0

13) 備考 1) 枝梗稲病度 (%) で比較した。カッコ内は指数比を示す。

1. Kinds
2. Inoculation date
3. 3 weeks after inoculation
4. Panicle branch blast
5. Neck blast
6. Total
7. October 24
8. Panicle branch blast
9. Neck blast
10. Total
11. Aichiasahi (Panicle completion period)
(milk ripe period)
(starch ripe period)
no inoculation
12. Tosan no. 38 (panicle completion period)
(milk ripe period)
(starch ripe period)
(yellow ripe period)
no inoculation
13. Remark 1) Degree of panicle branch desease (%) was compared.
Parenthesis indicates index ratio.

Infection of node at the neck of panicle had tendency to increase for inoculation at later period but were very few in Tosan no. 38 for inoculation at yellow ripe period. Examinations on number of infections are missing in some of Aichiasahi for inoculation at starch ripe period and in some of Tosan no. 38 for inoculation at yellow ripe period. These examinations were

cancelled because of early panicle death after development of desease. According to observations, infections of spikelet and panicle branch are characteristically large in both sections. From these experimental results, it is clear that panicle branch is easily infected at later period after heading. Desease was not observed in standard without inoculation.

Table 31 shows the degree of panicle branch desease. According to the table, the degree of panicle branch desease in Aichiasahi on 3 weeks after inoculation was 13.7 % for panicle completion period, 28.6 % for milk ripe period and 61.6 % for starch ripe period. The deseases increase significantly for later period inoculation after heading. Tosan no. 38 had an entirely similar tendency in the development of deseases. They reached 72 % for inoculation at yellow ripe period. Many deseases were developed on October 24 right before harvest.

Neck blast is slightly fewer than panicle branch blast or about the same (end of October). On three weeks after inoculation, the deseases tend to develop more for inoculation at later period but less at the end of October right before harvest in both kinds. This reversal in the tendency of desease development is an interesting fact compared with the tendency of panicle branch blast. The effects on the death of panicles are different depending on the infected parts and periods.

From the proportion of panicle branch blast among total panicle blasts, the following can be said. 1) Panicle branch blast was about half of total panicle blasts when inoculated soon after heading. However, panicle branch blast was high in inoculation after starch ripe period and 67 % in Aichiasahi for inoculation on September 28. Whereas it was 92 % in Tosan no. 38 for inoculation on October 4. 2) The proportion of panicle branch blast and neck blast was different depending on the period of examination even if they were inoculated at the same period. The ratio of neck blast becomes high with increase in elapsed days after inoculation. Conversely, the ratio of panicle branch blast becomes low. The reasons are the following. Neck blast does not cause the death of panicles in short period of days after infection and the expanded lesion results in a white panicle. The panicle branch blast invades the node at the neck of panicle due to death of panicle and is counted as neck blast at the end of October.

Variation of panicle branch blast inoculated at different period after heading is shown in figure 7.

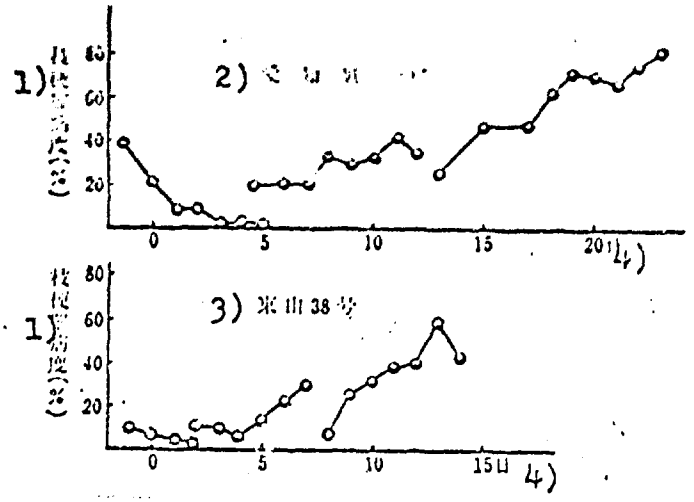

Fig. 7.—Variation of panicle branch blast inoculated at different period after heading. Upper, variety Aichiasahi; Lower, variety Tosan 38.

1. Panicle branch blast (%)
2. Aichiasahi
3. Tosan no. 38
4. Days

Aichiasahi in Fig. 7 shows that panicles have most deseases during heading and become more and more resistant as the days elapse after heading. Conversely, the panicles inoculated at milk ripe period and starch ripe period had more deseases with elapsed days after heading. Tosan no. 38 had similar tendency as above.

Weather during the experiments (see fig. 6) was low temperature of average 25°C at the end of August and warm temperature with clear sky at the beginning of September. According to weather bureau, autumn weather visited earlier in September. There was plenty rain after the middle of September and typhoon on September 10 and 26.

As can be seen from this weather, inoculations on Sept. 10 and Sept. 28 correspond to the day right after typhoon. Therefore, one of the reasons for development of many deseases in old panicles in this experiment was that the infection was promoted by wounds from typhoon and the desease has smoothly progressed by a long rain in second half of September. Deseases in panicle completion period were many in young panicles whereas in milk ripe and starch ripe period, the deseases were many in

nowhat old panicles. This seems to confirm the relation between desease and wound from wind as described in chapter IV. From this experiment, infection and death of panicle branch occur about 20 days after the heading of node at the neck of panicle in Aichiasahi (23-24 days after panicle completion period). and about 30 days after the heading in Tosan no. 38 (33-34 days after panicle completion period).

b. Infection at Later Period and Damage of Grain

Table 32 shows the relation between inoculation period and weight of rice ear, spikelet, and weight of 1000 grains of unpolished rice.

Table 32

Inoculation at Later Period and Grains

1) 品種	2) 接種時期	3) 接種月日	4) 葉稍病斑程度	5) 1穂重	6) 稔歩合	7) 精玄米1,000粒重
			%	g	%	g
8) 愛知旭	無接種	—	0	2.0	7.6	21.44
	穂揃期	9.11	10.1	1.6	22.2	20.64
	乳熟期	9.18	31.6	1.6	26.2	20.24
	糊熟期	9.28	68.2	1.6	18.9	20.15
9) 東山38号	無接種	—	0	1.9	6.8	20.24
	穂揃期	9. 6	14.0	1.6	16.3	20.03
	乳熟期	9.11	18.3	1.8	18.6	19.53
	糊熟期	9.18	49.2	1.8	14.6	19.07

10) 備考 1) 愛知旭は9月9～10日，東山38号は9月5～6日に穂首節の抽出した穂だけについて調査した。

1. Kinds
2. Inoculation period
3. Inoculation date
4. Degree of panicle branch desease
5. Weight of one ear of rice
6. Spikelet ratio
7. Weight of 1,000 grains of unpolished rice
8. Aichiasahi no inoculation
   panicle completion period
   milk ripe period
   starch ripe period

2. Tosan no. 38 no inoculation
panicle completion period
milk ripe period
starch ripe period

10. Remark 1) Ears of Aichiasahi whose node at the neck of panicle was formed on 9-10th of September were examined and those of Tosan no. 38 whose node at the neck of panicle was formed on 5-6th of September were examined.

According to table 32, the desease in both kinds was the least when the panicles were inoculated at panicle completion period, medium at milk ripe period and the most at starch ripe period. No desease was observed in rice plant without inoculation. In Aichiasahi, 1) Weight of ear was 2.0 g without inoculation whereas it was 1.6 g in inoculated sections. In spite of a considerable number of deseases during starch ripe period, it was not light. 2) Spikelet ratio was 7.6 % in no inoculation section, 22.2 % in panicle completiin period and 26.2 % in milk ripe period and increases in proportion to development of desease. In starch ripe period, it was 18.9 % and did not increase in proportion to desease. 3) Weight of 1,000 grains of unpolished rice was 21.44 g in no inoculation section, 20.64 g in ear completion period, 20.24 g in milk ripe period and 20.15 g in starch ripe period. It was slightly lighter for inoculation at later period. Thus, the more the deseases are, the lighter it is. Tosan no. 38 had about the same tendency as above.

These results indicate that the blast bacilli invade in about 3 weeks after heading in Aichiasahi (weakly resistant) and about two weeks after heading in Tosan no. 38 (strong resistant) and cause the death of panicle branch. As a result, the quality is lowered. In the case of infection at later period, the weight of ear and spikelet ratio do not become worse in proportion to the degree of desease. This is probably due to a certain degree of grain formation prior to infection. The reason for decrease in weight of 1000 grains of unpolished rice by the later period inoculation is that they are invaded after the grains are formed to a certain degree and these grains are counted as unpolished rice.

VII Discussion

1. Development of Desease

The parts of initial infections in panicle branch blast are mainly spikelet, node of rachis and node of panicle branch. Sometimes, the infections can be seen in devoluted parts of each organ or the space between the nodes. The lesions extend

to the direction of lower parts from the infected parts. In other words, the lesions expand in the direction of node at the neck of panicle. If the lower parts of nodes were invaded due to "death of ear", the panicle branch located on upper part of node dies and the dead part of ear expands rapidly. The spikelet on panicle branch cease to ripen and produce incomplete rice. Expansion of lesion in young ear is very rapid and the deseas developed in young ear causes damages over the wide area. The damages are larger if the parts of initial infections are lower parts of ear. Deseases of spikelet are easily developed and the number of deseases is also large. Thus, the damage due to spikelet infection occupies more than half or most of total damage in panicle branch blast. "The death of ear" due to spikelet desease does not affect the entire primary panicle branch. However, the desease of spikelet located near the node at the neck of panicle causes neck blast. Similarly the desease of spikelet located near the node of rachis causes the rachis blast and inflict a large damage.

The machanism of desease development in panicle branch blast was investigated by Sawada (1927), Kitsui (1936), Ito et al (1937), Kono(1954) and Kono and Suzuki (1960) and node of rachis or node of panicle branch was considered to be the parts of initial infection.

Sawada (1927) has observed many infections on junction part. The reason is that existence of white hair makes dew drops adhere on junction part of ear. This also makes adherence of blast bacilli conidium and invasion of bacilli easy, thus creating the favorable environment for expansion of lesion. We have also observed the infection of node of rachis and node of panicle branch. In this part, the desease tends to advance rapidly in the direction of the node. This is in agreement with the view point of Sawada. Yoshii (1936) has investigated the method of blast bacilli invasion and reported that epidermis or leaves near the junction point in node of panicles are most easily invaded in agreement with the view point of Sawada. Ito et al (1937) observed the invasion of blast bacilli on junction part of panicle and reported that the bacilli invasion takes place on inside of junction part. Recently, Kono and Suzuki (1960) cut the ear before heading and inoculated. As a result, they observed bacilli invasion in many parts of ear. The parts of initially developed natural desease (25-30 days after heading) were mostly node of rachis and node of panicle branch in addition to leaves. The deseases on the space between the nodes were few.

We have similarly observed important role of node of rachis or node of panicle branch as a part of initial infection. However, above reports differ from our view point in that the

development of panicle branch blast based on spikelet infection are not mentioned in their report. Although Kuribayashi (1929) already reported that spikelet itself is easily invaded, no mention has been made on the fact that "death of ear" from spikelet blast causes the panicle branch blast.

2. Infection of Spikelet

When blast bacilli invade a young spikelet, the parts on lemma are easily penetrated and bacilli invade through membrane of external epidermis.

Suzuki (1934) studied penetration method of bacilli in spikelet and reported that conidium drops into glume in bloom or bacilli reach the spikelet through anther and probably invade glume or rice grain. Since the tissues in internal epidermis ofglume are soft, penetration seems to be easy. Also, bacilli of wheat mould are said to invade through internal epidermis (Pugh et al 1933) or anther (Goto et al 1953, Nishimon 1958). Therefore, this possibility exists in blast bacilli and Suzuki's theory is interesting from the standpoint of infection mechanism. However, the conidial dispersion is few in day time (Kuribayashi et al 1952) and the opening of glume in spikelet is done in short time during the day (Nagai 1949). We have also observed that spikelets are easily infected during the ripening period. Thus, the mechanism of spikelet blast infection can be explained by Suzuki's theory in many cases.

When blast bacilli invade leaves of rice plant and node at the neck of panicle, they penetrate through epidermis (Matsuura 1928, Suzukata et al 1931, Yoshii 1933 and 1934, Kamura 1940). Since glume is considered to be similar organ as leaves (Breda De Haan 1911), it is quite reasonable that the penetration of bacilli in glume is through epidermis. However, glume lacks special cellular tissues that silicify difficultly unlike leaves and has a soft internal epidermis. Thus, penetration and activity of bacilli after penetration including appearance of symptoms are somewhat different from the case of leaves.

When unripened spikelets are inoculated, the desease is rapidly developed and the symptoms start to appear in 72 hours after inoculation. Lesions on glume turn to grevish white color around the penetrated parts and expands. The dead parts are not clear in many cases but different from typical lesion on leaves. This grevish white lesion sometimes resembles the white lesion on leaves. In spikelet blast, a small brown lesion similar to brown type in leaf blast appears in addition to grevish white lesion.

At an advanced state of desease conidia are markedly increased on epidermis of diseased spikelet and panicle. Syol (1940) observed conidial formation in vascular bundles of dead leaves, but we have not observed conidial formation on tissues of glume in our experiment. Desease is easily developed in young spikelet and conidial formation seems to be rapid due to the fast advancement of desease. This conidium is considered to form a source of secondary infection in neck and panicle branch blasts at later period.

Tip of lemma in spikelet is easily invaded and the reason for this is as follows. 1) Lemma has wider surface area than palea and shoulder part is saddle shape with many hair. Therefore, the bacilli have a better opportunity to adhere near the tip of lemma. 2) Considering the structure of glume, moisture holes and stomata are more at the tip than other parts. The discharged liquid from moisture holes promote the formation of dew drops and germination and growth of bacilli. 3) There are many hairs at the tip and the large hairs seem to be easily damaged by the friction of spikelet. Therefore, the infection due to wound increases. 4) Internal epidermis or soft tissues are more at the tip than other parts. Although there may be no direct relationship between the development of soft tissues and difficulty of bacilli penetration, these parts provide a favorable environment for activity and spread of bacilli. 5) Many stomata at the tip mean the presence of biologically active tissues. Also, at the tip of spikelet, the contact of palea and lemma is not close and internal epidermis can be directly contacted to outside. Thus, the blast bacilli can easily invade through the tip.

In short, the tip provides a favorable conditions for penetration and spread of blast bacilli and promotes the infection and development of desease.

3. Wound and Panicle Branch Blast

The ear receives severe wounds from treatment of artificial wind, and the deseases of panicle branch blast increase. The degree in development of desease is closely related to the number of wounds and severe wounds cause more deseases. Suzukata et al (1931) and Sakamoto (1940) also reported that wounds make infection of leaf blast easier. Shimada (1937) reported that other parts of rice plant in addition to wounded parts are susceptible to desease in leaf blast. Ear treated by artificial wind may have the effects reported by Shimada, but this relationship was not found. It was frequently reported that panicle blast increases after typhoon (Yoshida 1943, Ohda et al 1954, Agihara et al 1958).

Our experimental results seem to prove these observations.

4. Period of Infection

The incubation period was somewhat longer in young materials and shorter in old materials. The development of desease was rapid in young spikelet, and symptom of desease appears in 3.5-4 days after inoculation. The incubation period was more or less different depending on the parts of panicle. The short incubation period in spikelet is due to presence of many soft tissues and rapid spread of bacilli. On the contrary, the long incubation period in node at the neck of panicle or node of rachis is due to presence of thick membrane tissues and slow pread of bacilli. Thus, it is difficult to observe symptom of desease at initial period in this case.

From investigation of natural infectious period at farm, we have observed that infection of panicle branch blast occurs not only at early period but also later period including starch ripe period.

5. Infections at Later Period and Damage

Infectious period of panicle branch blast was considered to be about the same period as neck blast judging from the effective period of medicinal prevention (Nakagawa et al 1954 and 1955, Yasu et al 1957, Mori et al 1958, Yamanaka et al 1958, Motohashi et al 1959, Kogawa et al 1960, Yamauchi et al 1960). In the farm where neck blasts are high, the infections of panicle branch blast and neck blast are numerous right after heading and the damage of ear increases due to expansion of lesion. Thus, the damage due to infection at later period was hidden and seemed to escape observation. Also the mild panicle branch blast was not included in the standard of desease examination (Prevention of plant epidemics section, Agricultural improvement Bureau, Agriculture and Forestry Department, 1956) and the milk panicle branch blast at later period was probably not recorded.

Agihara et al (1958) observed newly developed panicle branch blast in relatively later period of panicle growth. Uzi et al investigated the natural desease of Norin no. 6 and Asahi no. 2 and indicated the following. The proportion of desease by periods had the same tendency in both kinds, and the desease was rarely developed in two weeks after heading. Then, the deseases increase gradually and reach about 50 % in one month. The rest was the newly developed desease after typhoon of Sept. 30 and October 4. 1) In Norin no. 6, the newly developed deseases are many in 1-2 days after typhoon. This symptom of

desease seems to have suddenly appeared due to effect of typhoon. 2) In Asahi no. 2, the newly developed deseases are many in few days after typhoon and the infections seem to be promoted by typhoon but some doubts exist on this effect.

The incubation period of blast desease is different depending on the environment (Itsumi et al 1936, Imura has developed, the progress of desease can be temporily ceased. Thus, the panicle branch blast developed at later period can not be said with certainty to exclude the infections at early period. Expression of some doubts on the infections at later period by Agihara was probably due to the above consideration.

We have already reported summary of infection on node at the neck of panicle and occurrance of white ear at later period (1958). Here, we report that panicle branch was easily infected at later period (chapter VI), the incubation period (chapter V) was shorter in old panicle than in young panicle and wind causes damage in spikelet and panicle branch, thus promoting the development of panicle blast (chapter IV). From these view points, the development of deseases at later period in the experiments of Agihara et al (1958) seems to be due to infections prior and after the typhoon.

The degree of panicle branch desease due to infections at later period was high and the quality of the product was lowered. This agrees with the observations on damages by neck blast (Hori et al 1958) and panicle branch blast(Agihara et al 1958) developed at later period. Thus, panicle branch blast developed at later period should be closely watched.

VIII. Conclusion

The development of panicle branch desease is not simple. "Deathes of ear" from infection of spikelet are numeroud and infections at node of panicle branch or rarely at the space between the nodes are observed. Infection of spikelet mainly occurs at the tip and there is a reason to believe that this part is easily susceptible to desease even at later period. Also, it is characteristics of panicle branch blast that invasion of lesion at the node increases the damaged area in stepwise.

The panicle branch blast can be said to be ear blast ecologically but is developed later than neck blast. The reasons are the following. 1) Panicle branch is located on upper part compared with neck of ear and subjected to more wounds. 2) Among the parts of panicle spikelet lives until the last minute and can be subject to blast desease for long time. 3) Progress of panicle branch blast due to death of the part lengthens the

infectious period and time between infection and development of desease. Thus, determination of preventive period is not simple and for this reason, research on this subject has been carried on until today. Determination of preventive period of panicle branch blast was conducted and there is an example of effective prevention at later period. Before judging the results of agricultural medicinal spreading at farm, however, these facts should be considered together with weather conditions. Then, a more accurate judgement can be made on preventive period derived from the experimental results.

IX. Summary

This paper reports the development of panicle branch blast by inoculation and observations at farm. Since spikelets are considered to be important as an infected part of panicle branch blast, we have made pathological and anatomical observations on the details of invading blast bacilli in spikelt, and reported on the relationship between sounds and deseases, incubation period, infectious period and damage due to infection at later period.

1. Panicle branch blast is classified into followsing four categories, considering the mode of pathogenesis by inoculation and characteristics of infected parts. They are spikelet blast (including glume blast, I type), secondary panicle branch blast (II type), primary panicle branch blast (III type) and rachis blast (IV type). Secondary panicle branch blast is further classified into secondary panicle branch blast started from spikelet (II-a type) and secondary panicle branch blast started from node of panicle branch (II-b type). These classifications explain sufficiently the status of desease.

2. Lesions expand from the tip of panicle to lower part, i,e, to the direction of node at the neck of panicle. Thus, "death of ear" occurs and death on the parts of panicle increases. Due to "death of ear", spikelet infection causes panicle branch blast in many cases.

3. Panicle branch blast occurs from infection of nodes of rachis and panicle branch in addition to spikelet infection. Panicle branch blast can also occur from infection of the space between the nodes and devoluted part of each organ, but the ratio is very small.

4. The relation between infected parts and degree of panicle branch desease shows the following.

Infection on node of rachis> infection on node of panicle branch> spikelet infection for one infected part. The damage is

large in infection on node at the neck of panicle or rachis. When total damage of panicle branch blast was considered, the degree of damage is in the order of spikelet infection> infection at node of rachis > infection at node of panicle branch. This is influenced by number of infections in addition to expansion of lesions. Thus, when panicle branch blast is considered, pathogenesis of spikelet can not be ignored.

5. Spikelet deseases are more in lemma than palea, particularly near hair.

6. When pathogenesis of spikelet blast is analyzed, the tissues of lesion are dead or destroyed, and the desease of tissues becomes mild at the boundary of infected part and healthy part. Spread of mycelia is significant at internal epidermis of glume, medium at external epidermis and very slow at lower tissues.

7. In spikelet desease, conidium is formed on the deseased part of glume (including tip) and inside of glume. They are scattered on the surface of deseased part and concentrated on the internal epidermis of glume.

8. When young spikelets are inoculated, blast bacilli invade through epidermis of glume. Invasion of bacilli is easier at upper half of spikelet, specially from the shoulder of lemma to tip, The invasion is difficult at base part of spikelet.

9. Mycelia which have advanced to glume reach the lower tissues in 24 hours after inoculation and soft tissues and internal epidermis in 48 hours after inoculation. In 72 hours after inoculation, they spread in various tissues.

10. Extent of mycelial spreading in tissues depends on the part of glume. Thus lemma is more than lower part. Mycelial spread is active in the soft tissues of glume and small in the thick membrane tissues.

11. When rice plant is treated by 9 m per second and 12 m per second artificial wind for two and half hours or three hours, spikelets and panicle branches are severely wounded. Wounds of spikelet appear as elliptical shape lesion or irregularly circular lesion. Sometimes, the entire spikelets discolor to dark brown. Young spikelets are more easily damaged. Wounds of panicle branch appear along the lines of vascular bundles. The wounds are mild in young panicle and severe in old panicle. Wounds are also severe in treatment of strong wind and mild in treatment of weak wind.

13. Incubation period of panicle branch blast in glass room (26°C) is about 7.5-11 days and depends on more or less infectious period or the parts of initial infection. Generally, incubation period is long for infection at panicle completion period, medium for infection at milk ripe period and short for infection at starch ripe period. When each part of panicle is compared, the incubation period is short in spikelet, medium in node of panicle branch or node of rachis and long in node at the neck of panicle.

14. Natural infection of panicle branch blast occurs continuously over the long period from heading to starch ripe period. The most infectious period depends on the yearly environments but is not unusual that it could occur in starch ripe period.

15. Infection of panicle branch by inoculation can easily occur on 16-20 days (20-24 days from panicle completion period in Aichiasahi) after node at the neck of panicle is formed, or around 30 days (33-34 days from panicle completion period in Tosan no. 38), and results in the death of panicle. When incubation periods are added, infection occurs in 4 weeks or 5-6 weeks after the node at the neck of panicle was formed and results in the death of panicle branch.

16. When Aichiasahi and Tosan no. 38 are inoculated at panicle completion period, milk ripe period, starch ripe period and yellow ripe period, the panicle branch blast was developed more for inoculation at later period. When the development of panicle branch blast is compared with neck blast, inoculation at early period produces about the same degree in both cases but inoculation at later period produces more panicle branch blast. The increased number of panicle branch blasts at later period seems to be due to effects of typhoon.

17. Panicle branch blast was not too high for inoculation at panicle completion period but the decrease in the weight of ear and increase in spikelet ratio are observed. Although panicle branch desease was high for inoculation at later period, the decrease in weight of ear and increase in spikelet ratio were not significant. However, the weight of 1,000 grains of unpolished rice tends to be light as the inoculation was conducted at later period. Thus, it seems reasonable to conclude that damage is also done by infection at later period.

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